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Management of Esophageal Hiatus Hernia Syndrome and Associated Abnormalities with Balanced Operations**,*

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In previous publications[1,2,3] we defined the various types of hiatal hernia and we listed them as follows: (1) esophageal hiatal hernia or the so-called sliding or short esophageal type (Fig. 1), (2) para-esophageal, para-hiatal or rolling type of hernia, which in our experience is rare (Fig. 4), (3) hiatal hernia en masse which is in reality a combination of hiatal hernia and para-hiatal hernia and which is more common (Figs. 5 and 7), and lastly (4) the extremely rare variety known as true congenitally short esophagus. This may be an inordinate variation of sliding hernia (Fig. 8) beginning early in life or it may represent an arrest of gastric descent. In all of these variations, however, the common denominator is a cephalad displacement of the cardia of the stomach above the diaphragm and a loss of the esophagogastric angle or the so-called angle of His (plica cardiaca). This angle is preserved normally by the phreno-esophageal fascia and the right crus of the diaphragm (Table I).

Esophageal Hiatus Hernia Syndrome

Hiatus hernia syndrome must be looked upon not only as a cephalad displacement of the cardia, but also as a malfunction of several foregut derivatives which behave as a physiologic unit. The foregut parts of the alimentary canal are nourished mainly by blood from the celiac axis and the portal vein, innervated by the vagi and splanchnics, and concerned primarily with digestion. Therefore, one unit affects another in response to normal and abnormal stimuli. This is observed in the variability of pain distribution in gall stone colic or esophageal, gastric or pancreatic disease.[11]

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TABLE 1—SYMPTOMATIC HIATAL HERNIA (Esophageal Hiatal Hernia Syndrome)

Types	Total Number	Number Having Balanced Operation	Per Cent of Total Studied
All types	425	105	24.7
"Sliding"		67	15.7
Hiatal hernia "en masse"		30	7.0
Parahiatal		7	1.6
Congenitally short esophagus		1	.23

Their common blood supply provides for the mutual and variable distribution of blood as needed. Normally many arteriovenous shunts exist in the esophagus, liver, stomach, and pancreas and they are exaggerated under physiologic demands and in certain disease states, such as hepatic cirrhosis[5,6] and probably also in hiatal hernia. Tachypnea and dyspnea increase intrathoracic negative pressure and increase the differential in pressures which ordinarily exist between the pressure in the coronary (left gastric) vein and the esophageal plexus in the thorax. This fact also increases venous hepatofugal flow in cirrhosis, hiatal hernia, and other diseases. Distension of the stomach increases portal pressure although we believe this to be purely mechanical since the phenomenon can not be demonstrated with the stomach outside of the abdomen in the experimental animal. However, gastric distension in the herniated stomach does cause pressure effects on venous return.[4] Esophageal varices which are noted in cirrhosis due to outflow hepatic obstruction are, we believe, really arterio-venous plexuses. This same phenomenon may explain bleeding in hiatal hernia without esophagitis.

The etiology of esophagitis in hiatal hernia is uncertain. However, we believe that the disease is due to two main causes: a reflux of highly acid gastric juice, and local tissue susceptibility. The clinical history

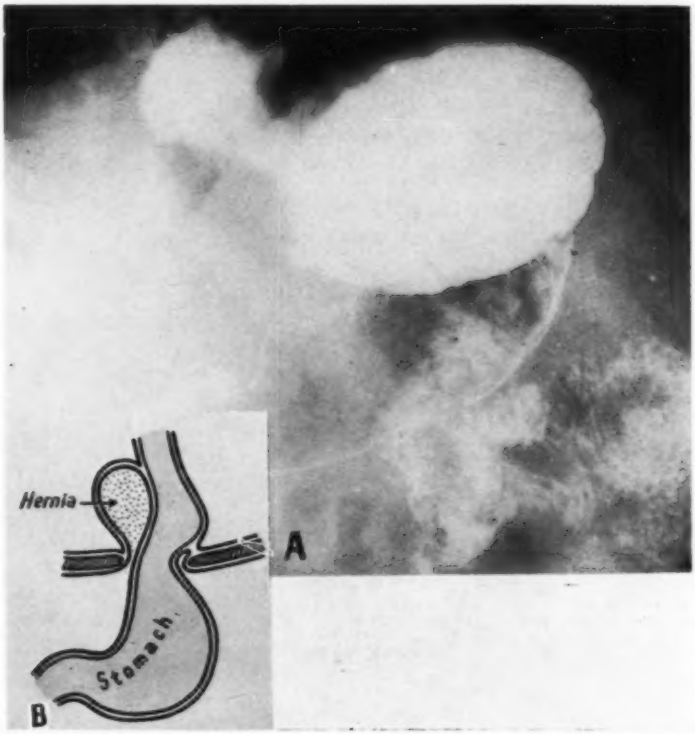

FIGURE 1: M.W., 41-year-old man with esophagitis and a sliding type esophageal hiatal hernia. (A) Preoperative postero-anterior x-ray photo following barium swallow. The cardia is above the diaphragm and the cardio-esophageal angle is lost. (B) Diaphragm illustrating the findings at surgery.

of pyrosis and regurgitation attests to this reflux as do cinefluorographic studies with barium (Fig. 3). It is true that regurgitation may occur without a hiatal hernia, but it is surprising how rarely this phenomenon is observed on fluoroscopic examination even in the Trendelenburg position when the hiatus and the esophagogastric angle are normal. It is also noteworthy that after esophagectomy with intrathoracic esophagogastrostomy that patients complain of regurgitation even after surgically constructed valves. This is probably due in part to pressure differentials and led us to the conclusion that the stomach must be replaced below the diaphragm in the "balanced operation" to obtain good results (Fig. 10). No patient with esophagitis without a hiatal hernia has required surgery in this group of cases. Local tissue susceptibility to peptic digestion is variable and is due perhaps to the availability of protective mucous, as well as to some quality as yet unknown in the cells themselves. The constancy of regurgitation is more important than the degree of acidity.

Stomach acids vary with associated foregut disease and are increased in cirrhosis, biliary tract disease, inflammatory and certain neoplastic diseases of the pancreas, and also in pulmonary diseases, particularly in emphysema and in tuberculosis. Duodenal ulcer is more frequent after a surgically made porta-caval shunt, which is in fact an exaggeration of the compensatory shunts found in advanced cirrhosis.[10]

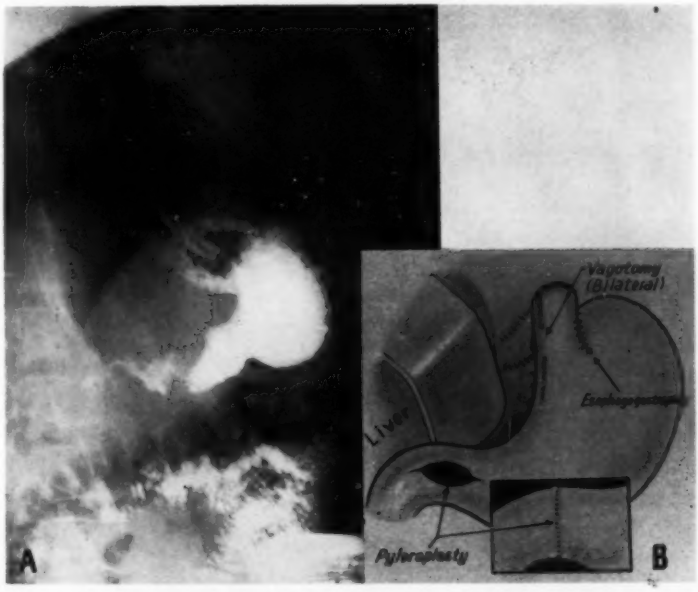

FIGURE 2: (A) Postoperative lateral x-ray film of patient shown in Fig. 1. The stomach has been restored to its normal position after "balanced operation." The esophagus may be seen through the gas bubble in the stomach (Magenblase). Note that the esophagogastric angle has been adequately restored by esophagogastropexy, and the pyloric opening is wide following pyloroplasty. (B) Diagram illustrating the completed "balanced operations" as performed in this case. The right crus of the diaphragm has been repaired and covered with a flap of phrenoesophageal fascia.

The causes of ulcerations in the stomach and duodenum have been the subject of a vast multitude of articles and investigations and as yet no clear-cut answer is available. However, it is of interest to note that in roughly 47 per cent of our cases the pathologists have found duodenitis, gastritis, or both, in varying degrees in the anterior third of the pylorus (which we remove routinely in our pyloroplasties). The association of gall bladder disease and pancreatic disease is well known, but perhaps less well known is the fact that pancreatic disease frequently results in a chronic or recurrent type of inflammation with scarring which is not unlike the process in cirrhosis and which presents a confused clinical picture associated with severe epigastric pain. Pancreatic juice under pressure may activate amylase which can be transported by hepatic venous capillaries as well as by the lymph channels of the liver and the pancreas. Cirrhosis of the pancreas which is not unlike nutritional cirrhosis of the liver has been described.[8] Apparently the pancreas goes through the same preliminary stage of fatty infiltration which is seen in cirrhosis and which definitely resembles nutritional cirrhosis of the liver.

All of the above relationships may explain the association of hiatal hernia with esophagitis, epiphrenic diverticulum, lower esophageal ring

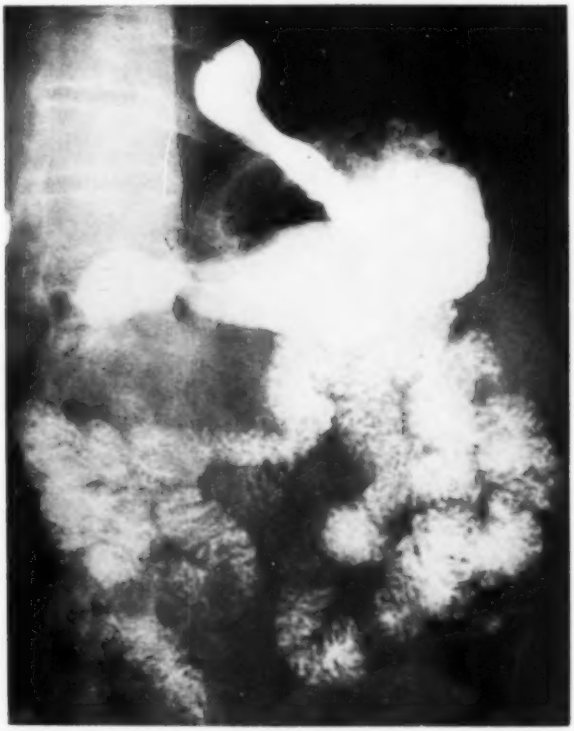

FIGURE 3: Mr. E.W., age 49. Preoperative film showing esophageal hiatal hernia of the so-called sliding type. There is a high degree of esophageal regurgitation, with a loss of the normal cardioesophageal angle. Considerable pylorospasm was present.

TABLE 2—ASSOCIATED LESIONS (ONE OR MORE) OF FOREGUT DERIVATIVES IN 105 PATIENTS HAVING BALANCED OPERATIONS

Type	No.	per cent	Operations Done	Results	Mortality No.	Mortality per cent
Esophagus						
Esophagitis	95	90.47	Balanced	Excellent*[1-4]	none	
Stenosis	3	2.8	Balanced & Heller op.	Excellent	none	
Epiphrenic diverticulitis	3	2.8	Balanced & diverticulectomy (thoraco & abdominal)	2 Excellent 1 Good	none	
Stomach						
Gastritis (biopsy)	32	30.47	Balanced	Excellent	none	
Ulcer	4	3.8	Balanced & gastric resection	Excellent	none	
Post-gastrectomy ulcer	1	0.95	Balanced	Excellent	none	
Large lesser curvature ulcer	1	0.95	Balanced & excision of ulcer	Excellent	none	
Duodenum						
Duodenitis (biopsy)	49	46.6	Balanced	Excellent	none	
Ulcer (active)	6	5.7	Balanced & gastric resection	Excellent	none	
Ulcer (inactive)	14	13.3	Balanced	Excellent	none	
Diverticulosis	1	0.95	Balanced	Excellent	none	
Liver						
Hemangioma	1	0.95	Balanced & excision of hemangioma	Excellent	none	
Cirrhosis with varices	2	1.9	Balanced	Good		
			Balanced & transesophageal ligation (thoraco-abdominal)	Poor	1	50
Gall bladder						
Stones	22	20.9	Balanced & cholecystectomy	Excellent	none	
Previous cholecystectomy	8	7.6	Balanced	Good	none	
Cholesterolosis	1	0.95	Balanced & cholecystectomy	Excellent	none	
Gall bladder & common duct stones	4	3.8	Balanced, cholecystectomy & choledochostomy	Excellent	none	
Common bile duct						
Strictures	3	2.8	Balanced & repair of duct	Good	none	
Fibrosis	1	0.95	Balanced & Roux Y choledocho-jejunostomy	Good	none	
Exploration	4	3.8	Balanced & sphincterotomy	Excellent	none	
Stones	4	3.8	Balanced & choledochostomy			
Pancreas						
Pancreatitis recurrent	5	4.7	Balanced, cholecystectomy & sphrincterotomy	Good	none	
Mal rotation with partial duodenal obstruction	1	0.95	Balanced, cholecystectomy & division of duodenal bands	Excellent	none	

*[1]1 patient had small incisional hernia 1 year after surgery; [2]1 partial recurrence, no symptoms; [3]2 patients have symptoms but no recurrence of hernia or esophagitis; [4]Eight patients had transient postoperative distention which responded to d-pentothenyl alcohol (Slopan). Diarrhea occurred in four, but required no specific medication; "dumping syndrome" was noted in three and was relieved by frequent feedings. Results are graded as follows: Excellent—no symptoms; good—vague occasional complaints but no recurrence; poor—no improvement, recurrence or death.

(Schatzki-Ingelfinger), duodenal or gastric ulcer, biliary tract disease, cirrhosis, and pancreatitis. A common type of mucous gland secretion in the derivatives of the foregut may account for some correlated diseases because when mucous in these organs is thick and viscid as in mucoviscidosis there is an association of bronchiectasis and fibrocystic disease of the pancreas, and occasionally the common duct may be obstructed by thick viscid mucous in the newborn. This is also true in Kartagener's syndrome. The association of tuberculosis and duodenal ulcer; pulmonary emphysema and gastric/duodenal ulcer; esophagitis and gastric/duodenal ulcer; cirrhosis of the liver and duodenal and gastric ulcer; gall

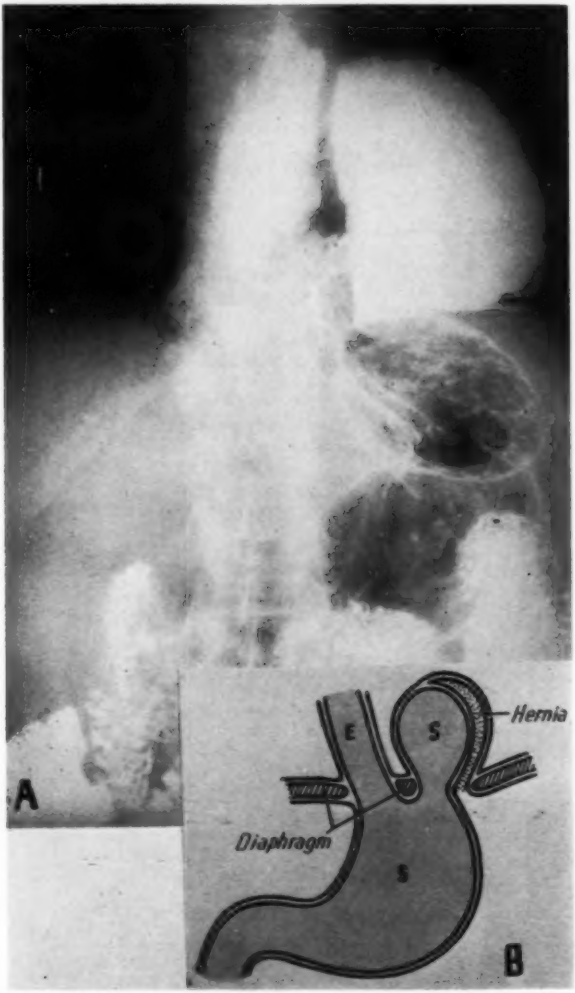

FIGURE 4: Mrs. M.S., age 64. (A) Barium swallow demonstrating a para-hiatal hernia. Note that the cardio-esophageal angle is below the diaphragm, but that the fundus comes up high along the side of the esophagus. This patient was admitted because of bleeding from associated esophagitis. This is not so common in the parahiatal type. (B) Diagram illustrating the condition found at operation.

stones and recurrent pancreatitis, may be due to mucin too thick to reach the surface or too thin to protect the embryologically related mucous membranes.

Symptoms In Hiatal Hernia Syndrome

Esophageal hiatal hernia may be asymptomatic. On the other hand the history of patients with associated esophagitis and/or other diseases of foregut derivatives is usually one replete with a long story of one or more of the following complaints: postprandial epigastric pain

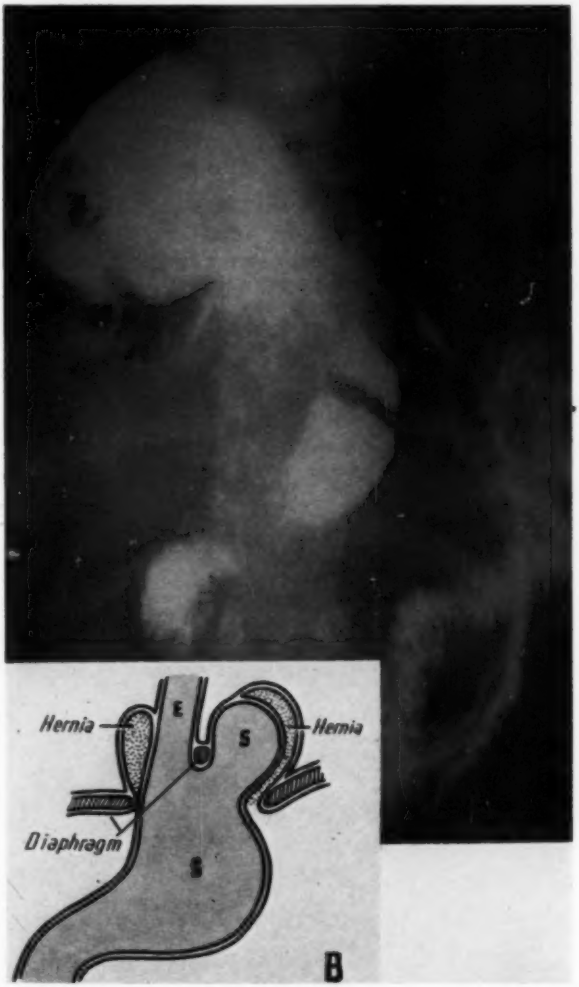

FIGURE 5: Miss E.S., 45-year-old woman with an enormous hiatal hernia en masse. (A) Preoperative film showing almost all of the stomach on the right side of the mediastinum and a loop of colon on the left. This patient had severe anginoid attacks, but came to the hospital because of hematemesis. (B) Diagram illustrating the type hernia which was found at operation. The colon is not depicted in the diagram, and much more of the stomach had extended upward than is shown (E=esophagus, S=stomach).

and discomfort, substernal burning (heartburn), dysphagia, pyrosis, dyspnea, regurgitation, aspiration with cough (particularly on reclining), vomiting, hematemesis, or general weakness due to profound anemia caused by easily discernable bleeding from the esophagus, stomach or duodenum; or occult hemorrhage from these organs which is unnoticed by patient and physician alike; also anginoid attacks which may closely resemble coronary disease, especially when the hernia is more medial or toward the right chest (Fig. 5). The pain is variable and often difficult to categorize. Many patients complain of discomfort behind the right ear, probably referred from the right crus by the phrenic nerve through the third cervical ramus to the great auricular nerve. We believe that the multiplicity of symptoms is due to the fact that malfunction is present in more than one foregut unit. This even applies to the post-cholecystectomy syndrome.

Progress of the Disease and Indications for Surgical Intervention

All of the patients herein reported have had a prolonged period of medical management without benefit, some for as long as 15 years. It has been our observation that the average person who has an uncomplicated

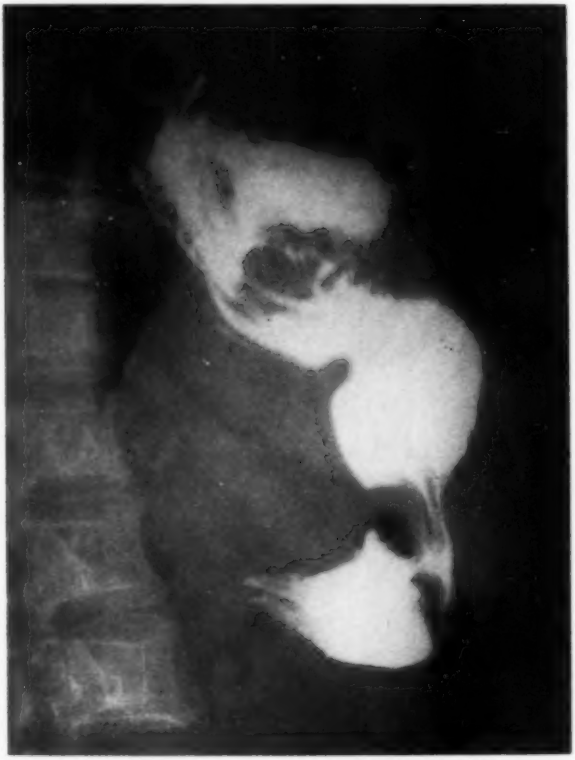

FIGURE 6: Postoperative lateral x-ray film of patient shown in Fig. 5, showing partial recurrence following severe wound infection in which a portion of the suture line gave way (see text).

hiatus hernia is relieved by diet and proper medication. This is not true of patients with complicated or persistent esophagitis.

Moreover, many of these patients had one or many operative procedures in an effort to get relief before consulting us. These included repeated esophageal dilatations, operations on the gall bladder, bile ducts, stomach, duodenum, ampullary sphincterotomy, cardiomyotomy (Heller), gynecologic procedures and others.

During the past nine years, we have studied 425 patients with symptomatic hiatal hernia. All had symptoms which were severe enough to warrant hospitalization, but only 105 (24.7 per cent) required surgery. The indications were as follows:

(a) Persistence and progression of symptoms in spite of adequate medical management.
(b) Complications such as stenosis, bleeding, or perforation.
(c) Associated lesions for which surgery is indicated, particularly in the esophagus, stomach, duodenum, pancreas, gall bladder and bile ducts.

We wish to emphasize the fact, however, that every patient should receive an adequate trial on medical management before any operative procedure is done. If there is associated disease for which surgery is needed, even though the hiatal hernia is thought to be asymptomatic, it should be repaired at the time and a balanced operation should be done.

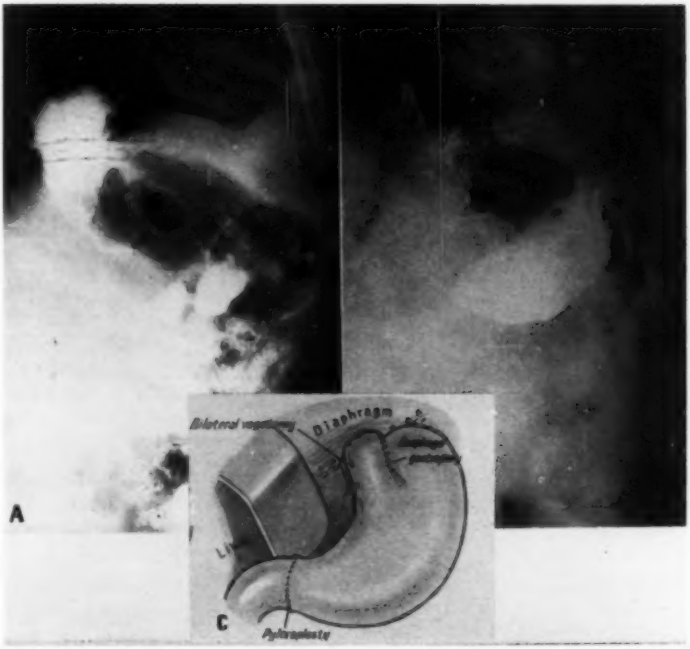

FIGURE 7: J.E., a 51-year-old man who came to the Marion County General Hospital exsanguinated from gastric hemorrhage. (A) Lateral x-ray showing a large hiatal hernia en masse. The arrow points to a wide shallow ulcer in the cardiac end of the stomach on the lesser curvature. The ulcer was excised and "balanced operations" performed. (B) Postoperative film. (C) Diagram illustrating the type procedure done in this case. The result was excellent.

This is necessary because it is often difficult to determine whether the companion disease, the hiatal insufficiency, or both, are the cause of persistent symptoms. Therefore we use the term "hiatus hernia syndrome."

Surgical Procedure

In attempting to relieve patients referred to us with hiatal hernia and esophagitis or associated foregut derivative disease we tried indi-

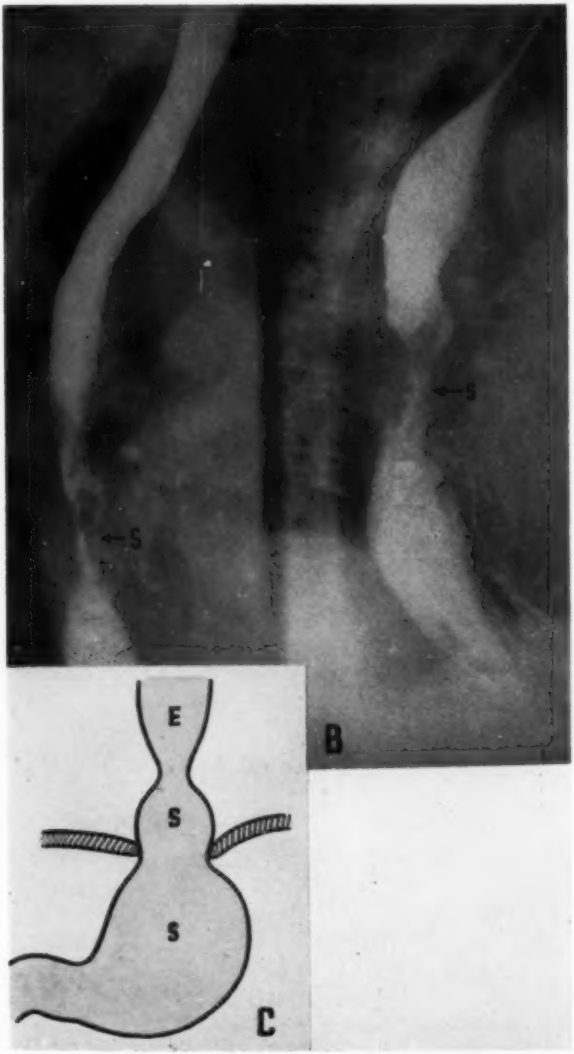

FIGURE 8: 27-year-old woman with so-called congenitally short esophagus with stricture. The patient also has scoliosis. (A and B) Note the high position of the stomach, together with a complete loss of cardioesophageal angle, and also the stricture "S" in the lower end of the esophagus. (C) Diagram illustrating the conditions found at the time of surgery (E=esophagus, S=stomach).

vidual procedures including radical resections as advocated by many authorities, and these have been referred to in our previous publications.[1,2,3] However, we were unable to obtain consistently good results. In some instances radical operations such as total gastrectomy or the interposition of a loop of jejunum or colon between the esophagus and the stomach were done, but many patients did not remain asymptomatic. This does not mean that the patients did not recover, but rather that we often substituted one type of complaint for another.

If we assume that symptomatic hiatal hernia is a concomitant malfunction of several derivatives of the foregut which behave as a complex physiological unit, then several operative procedures must be done in one stage to obtain a maximum benefit. We call these operations "balanced" because we replace the stomach in its normal position and re-establish the integrity of the hiatus. In addition, we attempt to produce a normal esophagogastric angle, reduce gastric acidity and facilitate gastric emptying, thereby reducing intragastric pressure (Figs. 2 and 7). This is accomplished as follows:

(1) The hernia is reduced.
(2) Vagotomy is done to reduce gastric secretion and to permit more complete descent of the esophagus.
(3) The hiatus is repaired by suturing the right crus, imbricating the sac in large hernias, and suturing the infolded sac to the under surface of the diaphragm.
(4) Esophagogastropexy is performed.
(5) A pyloroplasty is carried out in which the anterior third of the pylorus is removed.
(6) Associated lesions are corrected (Table 2 and Fig. 9).

These operations were done through an abdominal approach with five exceptions, even though previous to our present practice we did many of our repairs transthoracically. One patient with a true congenitally short esophagus type of deformity had esophagitis in her early youth (Fig. 8). In this case we mobilized the esophagus up to the arch of the aorta by transthoracic approach. This enabled us to replace the stomach into the abdominal cavity and to proceed with the balanced operation (and esophagoplasty). We obtained a satisfactory result and the esophagitis has improved remarkably. Three others had epiphrenic diverticula which required a combined approach, and one had bleeding esophageal varices due to nutritional cirrhosis.

The stomach must be replaced within the abdominal cavity. Experiments in animals have shown that the stomach in the chest is likely to produce gastric reflux whether or not a sphincteric mechanism is present;[6] and in a study of 59 cases of esophageal resection for carcinoma with thoracic esophagogastrostomy we have found that reflux is a problem in many cases.[7] The esophagogastric junction is inspected carefully for abnormalities because the passageway to the stomach must be unimpeded. We have had to do cardiomyomotomy in only three patients although many of our cases have had previous esophageal dilatations. For the most part it is our opinion that cardiospasm is largely corrected when the reflux stops and the inflammation and swelling within the esophagus recedes.

A reduction in the amount and acidity of gastric juice is desirable because we believe that high acidity is more likely to induce esophagitis than normal acidity or alkalinity. Therefore we do a bilateral vagotomy. However, esophagitis may occur when alkaline digestive juices regurgitate as in esophago-duodenostomy after total gastric resection. It is the continuous reflux of digestive material day and night which incites esophageal irritation as well as the high acidity.

The angle at which the esophagus enters the stomach must be made acute so that regurgitations will be hindered, and this is done by the esophagogastropexy. At the same time the egress of gastric contents through a widely open pylorus must be established so that pylorospasm will be avoided and intragastric pressure reduced. Lastly, associated "foregut" disease should be corrected if the patient is to be restored to normalcy (Table 2 and Fig. 9).

Statistical Survey

We have now performed one stage balanced operations for all types of hiatal hernia in 105 consecutive cases during the past nine years. The ages of the patients varied from 28 to 67 years. The average age for hiatal hernia was 48 years; whereas for hiatal hernia en masse it was 56 years. Forty-four per cent of our patients were women and 56 per cent were men. The preponderance of men was in the group of esophageal hiatus hernia, whereas the preponderance in women was in the group desig-

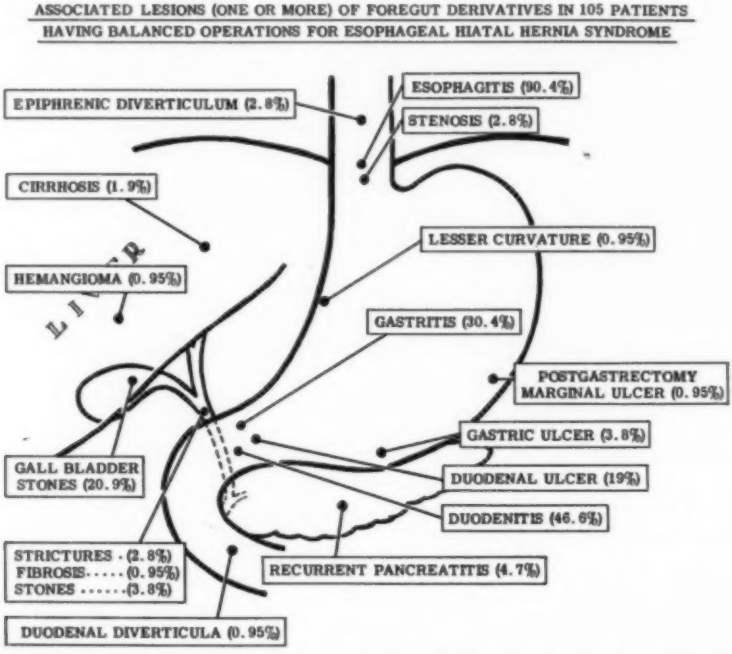

FIGURE 9: Diagram showing the associated lesions (one or more) of foregut derivatives in 105 patients having "balanced operations" for esophageal hiatal hernia syndrome.

nated hiatal hernia en masse. All patients had been on medical management from two to 15 years, the average being six years. Many had unrelated operations before the correct diagnosis was made. Three had gastrostomies and one a posterior gastroenterostomy, and the three referred to lived on gastrostomy feedings for one, one and a half, and two years, respectively. Esophageal dilatations were done in 14 patients. Five of these were reported as having a very tight stenosis refractory to dilatation, and yet cardiomyomotomy was not done in these, but was necessary in two cases of sliding hiatus hernia and the one case which had a true congenitally short esophagus.

The diagnosis was established in every case on clinical evidence, repeated fluoroscopic and x-ray examinations, in addition to other tests such as stomach acids, repeated stool examinations for occult blood to explain the secondary anemia. Esophagoscopy was done in many of the cases operated upon during the past four years.

Results

Our results have been almost uniformly good. The follow-up extends over a period from six months to nine years. There was one partial recurrence without symptoms which was discovered on routine post-operative x-ray examination. This 45 year-old woman had a large hiatal hernia en masse. She developed a post-operative wound infection with partial dehiscence of the abdominal incision as well as the antero-lateral por-

FIGURE 10: W.B., a 36-year-old man, came to the Marion County General Hospital because of severe peptic esophagitis with ulceration found on esophagoscopy. He had been in many hospitals prior to his admission and had had the following successive surgical procedures: (1) a 50% gastrectomy, (2) a revision of the gastrectomy in which 40% more of the stomach was removed leaving only approximately 10% of the fundus, (3) a bilateral transthoracic vagotomy with imbrication of the hiatal hernial sac and closure of the crus. Study of this patient by x-ray examination, fluoroscopy and by esophagoscopy showed constant regurgitation into the lower end of the esophagus. The gastric acids were high. This case clearly illustrates that relief from esophagitis in hiatal hernia must include a restoration of the normal esophagogastric angle and replacement of the stomach within the abdomen. S on the left= stomach above diaphragm. S on right=remaining portion of fundus. Arrow indicates site of gastro-jejunostomy. Right crus repaired and esophagogastropexy done through abdominal approach.

tion of the diaphragmatic repair (Fig. 6). This is the only known instance of recurrence in our series.

Two still have complaints. One of these had eight previous operations and lived on gastrostomy feedings for two years prior to our operation. She became completely well following her balanced operations, which were done three years ago. She was brought into the Marion County General Hospital on November 14, 1959 with all the symptoms and signs of grand mal. At this time esophagoscopy was done and the esophagus was found to be perfectly normal and stomach acids were within normal limits. The patient had a convulsion on the ward and injured her right shoulder. There were no gastro-intestinal complaints and she had gained 31 pounds. The second patient is also one who had eight previous operations, including a gastrostomy for feeding. This woman was improving rapidly and had gained some 20 pounds. However, she complained of vague abdominal pain and reentered the hospital where she had esophagoscopy and no residual esophagitis was found. She was placed under the care of a neuropsychiatrist who ascribed her symptoms as "defensive" due to incompatibility with her mother-in-law. When last heard from in February, 1960, she had improved.

There was one death in a 34 year-old man with nutritional cirrhosis of the liver and bleeding esophageal varices as well as a large sliding type hiatal hernia. A transthoracic transdiaphragmatic operation was done consisting of intraesophageal ligation of the varices and balanced operations. He developed an esophageal fistula at the site of the esophagotomy necessitating gastrostomy for feeding. He died seven weeks after surgery of hepatic insufficiency.

SUMMARY

Hiatal hernia syndrome is due to a malfunction of several of the derivatives of the foregut, as well as a cephalad displacement of the stomach. Therefore, we frequently encounter abnormalities of the esophagus, stomach, duodenum, gallbladder, bile ducts, pancreas, and even the bronchi and lungs.

Most patients with hiatus hernia syndrome may be treated successfully by careful medical management. However, roughly one-fourth will require surgery. These are the patients who have a persistence and progression of symptoms, in spite of adequate medical care, or who have esophageal complications, such as stenosis, bleeding, or perforation, or associated lesions of foregut derivatives.

If surgery is indicated for any of the above reasons, or for unrelated disease, a balanced operation should be done.

The steps of the operation and the reasons for its performance are as follows.

1. The hernia is reduced and prevented from recurring. (Step 3)
2. Bilateral vagotomy is done to reduce the amount of acidity of gastric secretion and to permit more complete descent of the esophagus.
3. The hiatus is repaired by suturing the right crus, imbrecating the sac (in large hernias), and suturing the phrenoesophageal ligament to the crus and the infolded sac to the under surface of the diaphragm.
4. Esophagogastropexy is performed to restore or exaggerate the esophago-gastric angle (angle of HIS) (plic cardiaca).
5. Pyloroplasty is carried out, in which the anterior third of the pylorus is removed, thereby permitting ready egress of gastric contents and relieving intragastric pressure.
6. Associated lesions are corrected.

This procedure has now been done in over 105 patients, with almost uniformly good results. All patients have been followed from six months to nine years, with an average of about four years. We have had only one known recurrence of herniation. No patients have required dilatations or other treatments of the esophagus following the balanced operations.

RESUMEN

El sindrome de la hernia hiatal se debe a una disfunción de varios derivados del canal digestivo asi como a un desplazamiento cefálico del estómago. Por tanto, frecuentemente encontramos anomalías del esófago, del estómago, el duodeno, vesícula, vias biliares, pancreas y aun de los bronquios y de los pulmones.

La mayoría de los enfermos con hernia hiatal pueden tratarse con éxito con procedimientos médicos cuidadosos. Sin embargo aproximadamente una cuarta parte de ellos necesita la cirugía. Tales son los enfermos que tienen persistencia y empeoramiento de los síntomas a pesar del cuidadoso tratamiento médico o los que tienen complicaciones esofágicas como la estenosis, hemorragia o perforación o son asociadas a las lesiones del tracto digestivo alto en sus derivaciones.

Si la cirugía está indicada por cualquiera de las razones antes señaladas o por una enfermedad sin relación con ella, debe hacerse una operación bien equilibrada.

Los pasos para la operación y las razones de su realización serían:

1. La hernia se reduce y se evita que recurra.
2. La vagotomía bilateral se hace para disminuir la acidez de la secreción gástrica y para permitir el descenso completo del esófago.
3. El hiato se prepara suturando el haz derecho, imbricando el saco (en las hernias grandes), y suturando el ligamento frenoesofágico al haz y en saco invaginado a la superficie inferior del diafragma.
4. La esofagogastropexia se lleva a cabo para restaurar o exagerar el ángulo esofagogástrico (ángulo de His) (Plic Cardiaca).
5. Se hace la piloroplastía en la que el tercio anterior del píloro se reseca permitiendo asi la salida facil del contenido gástrico y aligerando la presión intragástrica.
6. Se corrigen las lesiones asociadas.

Este procedimiento se ha realizado ahora en mas de 105 casos con resultados casi uniformemente buenos. Todos los enfermos se han observado de seis meses a nueve años con un término medio de cuatro años. Hemos tenido solo una recurrencia conocida de la hernia. Ningún enfermo ha requerido dilataciones u otros tratamientos del esófago después de estas operaciones equilibradas.

RESUMÉ

Le syndrome de hernie hiatale est imputable à une malformation de plusieurs des dérivés du tube embryonnaire, aussi bien qu'à un déplacement de l'estomac orienté vers la tête. C'est pourquoi nous rencontrons fréquemment des anomalies de l'estomac, de l'oesophage, du duodénum, de la vésicule biliaire, des canaux biliaires, du pancréas et même des bronches et des poumons.

La plupart des malades atteints de hernie hiatele peuvent être traités avec succès par une thérapeutique médicale soigneuse. Cependant, grosso-modo pour un quart d'entre eux, il est nécessaire d'avoir recours à une opération chirurgicale. Ce sont les malades qui ont une persistance et une progression des symptômes, malgré une surveillance médicale convenable, et qui ont des complications oesophagiennes, telles que sténose, hémorragies ou perforation, ou des lésions associées des dérivés du tube embryonnaire.

Si la chirurgie est indiquée pour chacune des raisons énumérées ci-dessus, ou pour toute autre complication, une opération équilibrée devrait être faite.

Les étapes de l'opération et les bases de sa pratique sont les suivantes:

1. La hernie est réduite et on tâche d'éviter sa rechute.
2. Une vagotomie bilatérale est réalisée pour réduire l'acidité de la sécrétion gastrique et permettre une descente plus complète de l'oesophage.
3. Le hiatus est réparé en suturant le pédicule droit, en imbriquant le sac (dans les hrnies volumineuses) et en suturant le ligament phrénoesophagien au pédicule, et au sac déplié à la surface inférieure du diaphragme.
4. Une oesophago-gastropexie est pratiquée pour réparer ou exagérer l'angle oesophago-gastrique (angle de His).
5. Une pyloroplastie est pratiquée, dans laquelle le tiers antérieur du pylore est enlevé, permettant ensuite l'élimination du contenu gastrique et soulageant la pression intragastrique.
6. Les lésions associées sont corrigées.

Cette technique a maintenant été pratiquée sur 105 malades, avec presque uniformément de bons résultats. Tous les malades ont été suivis de six à neuf ans, avec moyenne d'environ quatre ans. L'auteur n'a relevé qu'une seule rechute de la hernie. Aucun malade ne nécessita des dilatations ou d'autres traitements de l'oesophage après les opérations équilibrées.

ZUSAMMENFASSUNG

Das Syndom der Hiatus-Hernie ist die Folge einer Dysfunktion mehrerer Abkömmlinge des Vorderdarmes ebenso wie einer kopfwärts gerichteten Verlagerung des Magens. Wir stoßen daher häufig auf Mißbildungen von Speiseröhre, Magen, Zwölffingerdarm, Gallenblase, Gallengängen, Pankreas und sogar von seiten der Bronchien und Lungen.

Man kann die Mehrzahl der Kranken mit dem Syndrom der Hiatus-Hernie erfolgreich behandeln in Form einer sorgfältigen internistischen Betreuung. Ungefähr ein Viertel von ihnen wird jedoch operatives Vorgehen erforderlich machen; und zwar handelt es sich dabei um diejenigen Patienten, die trotz entsprechender internistischer Behandlung anhaltende und sich verstärkende Symptome aufweisen oder die Komplikationen von Seiten des Oesophagus bekommen wie z.B. Stenose, Blutung oder perforation oder damit verknüpfte Veränderungen im Bereich der Abkömmlinge des Vorderdarmes.

Besteht die Indikation zum Eingriff aus einem der erwähnten Gründe oder wegen einer damit nicht in Zusammenhang stehenden Erkrankung, sollte man eine wohl abgewogene Operation vornehmen.

Die Phasen der Operation und die Gründe ihrer Vornahme sind folgende:

1. Die Hernie wird beseitigt und ihr Rezidiv verhindert. (3 Phase)
2. Eine bilaterale Vagotomie erfolgt, um den Säuregehalt des Magensaftes zu verringern und ein vollständigeres Senken des Oesophagus zu ermöglichen.
3. Der Hiatus wird behoben durch Naht des rechten Schenkels, dachziegelartiges Übereinanderlegen des Sackes (Im Fall von großen Hernien) und Vernähung des lig. phrenoesophag. mit dem Schenkel und dem gefalteten Sack an der Zwerchfellunterfläche.
4. Eine Oesophago-Gastropexie wird ausgeführt, um die Winkelbildung zwischen Speiseröhre und Magen (His'scher Winkel oder plica cardiaca) wieder herzustellen oder wieder zu verstärken.
5. Eine Pylorusplastik wird vorgenommen, bei der das vordere Drittel des Pylorus entfernt wird, um damit den regulären Austritt des Mageninhaltes zu ermöglichen und den intragastrischen Druck herabzusetzen.
6. Mit dem Befund in Zusammenhang stehende Veränderungen werden korrigiert.

Diese Technik ist bis jetzt an über 105 Kranken angewandt worden und mit fast einheitlich guten Ergebnissen. Alle Patienten wurden 6 Monate bis 9 Jahre lang nachbeobachtet mit einer durchschnittlichen Zeit von etwa 4 Jahren. Es ist uns nur ein Fall von Hernien-Recidiv bekannt geworden. Bei keinem Kranken waren Dehnungen oder andere Behandlungen durch Oesophagus notwendig nach Ausführung der beschriebenen Operationen.

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Resectional Therapy for Pulmonary Tuberculosis at Sunmount, 1950 - 1957, 807 Cases

V. Resectional Therapy for Pulmonary Tuberculosis under Viomycin and Pyrazinamide Coverage

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In reviewing all patients who underwent resectional therapy for pulmonary tuberculosis at the Sunmount Veterans Administration Hospital between the years 1950 to 1957, a special study was made of the clinical course of those patients who had resectional therapy under viomycin (VIO) and pyrazinamide (PZA) coverage. Viomycin and PZA coverage for resection was elected when the patient had been demonstrated to be resistant to other chemotherapeutic regimens, and was instituted at various time intervals prior to surgery as shown in Table 1.

A total of 32 patients had resectional therapy under viomycin and PZA coverage. All 32 had cavitary disease; 28 were considered open-positive, and four open-negative. In this group of 32 patients, there was only one major post-operative complication, and that a non-tuberculous empyema with fistula due to hemolytic staphylococcus aureus. To date there has been only one clinical relapse among the 32 patients. This manifested itself by the appearance of a new cavity on the operated side more than one year following surgery. Six other patients have had a single isolated positive sputum at varying periods following surgery, but have shown no other manifestations of relapse. All 32 patients, incuding the one who showed relapse, are presently negative, but only four patients have been followed for a sufficiently long period to be classified as inactive.

It must be stressed that since all these resections were done between the years 1955 and 1957, the majority in 1956 and 1957, the follow-up is too short to allow conclusions about the eventual relapse rate. The absence of tuberculous complications, and the low incidence of complications in general, is striking when compared to the high incidence of

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TABLE 1—TIME RELATIONSHIP OF INSTITUTING VIOMYCIN AND PYRAZINAMIDE CHEMOTHERAPY WITH RESPECT TO RESECTIONAL THERAPY, AND PRE-OPERATIVE STATUS OF THE PATIENT

Time Relationship of instituting chemotherapy to surgery	Pre-op Status of Patient		
	Open-positive	Open-negative	Total
After Surgery: 1-7 days		1	1
At Surgery	6		6
Before Surgery: 1-7 days	2	2	4
8-14 days	6		6
15-21 days	2		2
21-28 days	1		1
1-2 mos.	6	1	7
2-3 mos.	4		4
3-5 mos.	1		1
Total	28	4	32

TABLE 2—COMPARISON OF OPEN-POSITIVE PATIENTS WHO HAD RESECTIONS UNDER VIOMYCIN AND PYRAZINAMIDE COVERAGE WITH OPEN-POSITIVE PATIENTS WHO HAD RESECTIONS UNDER INEFFECTIVE CHEMOTHERAPY COVERAGE

Drug Coverage	No. of Cases	Major Complications	Persistent Pos. Sputum	Relapses
VIO & PZA	28	1	0	1
Ineffective	28	13	11	4

tuberculous complications in patients who had resectional therapy under ineffective chemotherapy coverage. Table 2 compares resections in open-positive cases under VIO and PZA coverage with resections under ineffective chemotherapy as defined elsewhere.[1] By chance, the number of cases in each group is equal. The resections in the VIO and PZA group include eight pneumonectomies, 14 lobectomies, five segmental resections, two resections of a lobe plus a segment, two resections of a lobe plus a wedge, and one resection of a segment plus a wedge. Only five patients had resections carried out under a prior thoracoplasty; three of them had lobectomies, one a resection of a lobe plus a segment, and one a pneumonectomy. There was no subsequent thoracoplasty. The resections for the ineffective chemotherapy group have been listed elsewhere.[1]

Discussion

The absence of tuberculous complications post-operatively in the group of patients who had resections under viomycin and pyrazinamide coverage is striking. While the time of instituting this chemotherapy regimen prior to resection varies in the group of patients studied, it is our present arbitrary opinion that the optimal time for starting viomycin and pyrazinamide therapy is approximately two weeks prior to contemplated resectional therapy. In view of the immediate good results following surgery, the opinion that viomycin and pyrazinamide represent excellent secondary drug coverage for resectional therapy of pulmonary tuberculosis is confirmed by the experience at this hospital. In view of the studies of McLean[2] this should not be interpreted to mean that viomycin and pyrazinamide represent good long-term chemotherapy coverage in themselves, in the absence of resectional therapy. Data on the long-term follow-up of these patients is not available.

SUMMARY

Thirty-two patients who had resectional therapy for pulmonary tuberculosis under viomycin and pyrazinamide chemotherapy coverage have been reviewed. It is concluded that the experience at Sunmout testifies to the fact that viomycin and pyrazinamide represent satisfactory secondary chemotherapy coverage for pulmonary resections.

RESUMEN

Se revisan treinta y dos enfermos que se sujetaron a resección pulmonar por tuberculosis pulmonar bajo la protección de quimioterapia por Viomicina y Pirazinamida. Se concluye que la experiencia en Sunmount atestigua que la Viomicina y la Pirazinamida representan elementos de quimioterapia secundaria para la protección en casos de resección pulmonar.

RESUMÉ

L'auteur rapporte les observations de 32 malades qui ont subi un traitement chirurgical pour tuberculose pulmonaire sous couvert de Viomycine et de pyrazinamide. Il arrive à la conclusion que l'expérience de Sunmount apporte le témoignage que la viomycine et la pyrazinamide représentent une couverture chimiothérapique auxiliaire satisfaisante pour les résections pulmonaires.

ZUSAMMENFASSUNG

Bericht über 32 Kranke, die mit Resektion wegen Lungentuberkulose unter dem Schutz von Viomycin und Pyrazinamid behandelt wurden. Verfasser schliessen damit, daß die Erfahrungen des Sunmount Hospitals von der Tatsache zeugen, daß Viomycin und Pyrazinamid bei Lungenresektionen einen befriedigenden sekundären therapeutischen Schutz darstellen.

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A Method of Continuous or Intermittent Tracheobronchial and Pulmonary Infusion*

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In the course of investigations or treatment of bronchopulmonary disease it is often desirable to introduce agents directly into the bronchial tree. In humans this can be done by several methods. The most efficient non-surgical method is by bronchoscopy, and in this respect the authors of a recent publication utilize this procedure to introduce medicated lyophilized oil into tuberculous lesions.[1] Another similar method utilizes a soft rubber catheter introduced into the trachea under fluoroscopic control. This is essentially the method frequently used for instillation of radio-opaque material into the tracheo-bronchial tree, for bronchography. Any of the other methods used for bronchography, such as the supraglottic drip or the trans-tracheal method, may be used similarly for the introduction of medications into the bronchi.

The disadvantage of all these methods is that they are cumbersome and entail a great deal of co-operation on the part of the patient. They are also very ill suited for continuous therapy or for therapy requiring frequent instillations into the bronchi every few hours or daily.

For this reason the use of aerosol solutions, either with or without intermittent positive pressure breathing, has become widely accepted. The limitations of these methods are several. One is again patient co-operation, since the patient must be taught how to use the apparatus efficiently. Another is the amount of medication which can be introduced in this manner. Even discounting the quantities of material which may settle on buccal mucosa and pharynx and cause irritation in these areas, the amount which reaches the tracheo-bronchial mucosa is necessarily small and usually consists of about 1 cc. of solution three or four times a day — a considerable portion of which never reaches its intended destination.

The most efficient method would, of course, be tracheotomy, for with this, solutions to any amounts tolerated by the tracheo-bronchial tree could be introduced as desired throughout the day. Suction could be applied when necessary to remove unwanted secretions. The tolerance of the tracheo-bronchial tree for large quantities of fluid is remarkable. Nevertheless, even though tracheotomy is becoming more widespread and acceptable than formerly, it is still not favored as a routine method of therapy for the majority of cases.

The tracheal fenestration operation proposed by Rockey[2] meets some of the objections to most methods, but here again one exposes the patient to a considerable surgical operation and a period of wound healing is necessary which might easily interfere with the administration of certain solutions.

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As with straight tracheotomy, this method would not be desirable where a period of therapy covering only a few days or possibly a few weeks is contemplated.

In order to overcome these objections the author is attempting to introduce materials directly into the tracheo-bronchial tree via a plastic tube. The description of the method follows:

The skin of the neck for a considerable area over the trachea is cleansed and prepared, as for a surgical operation. Local anaesthesia is secured by skin infiltration for 1-2 per cent procaine and the anaesthesia is carried down to the trachea and between two cartilaginous rings into the lumen itself. This method of transtracheal anesthesia is a standard procedure in many institutions for bronchoscopy and bronchography and is practically devoid of risk.

A No. 14 gauge needle is now inserted through the prepared site into the trachea. Through this a plastic catheter is threaded until about one inch of the catheter is well in to the tracheal lumen. This should prevent the catheter from slipping out of the lumen with neck movements or with cough or swallowing. Some anesthetic is dribbled through the catheter so that its presence will be tolerated by the patient. (An aerosol of anesthetic solution given every three or four hours may also be of some value). The catheter can then be sealed and the seal opened for medication to be administered as often as necessary during the day or else if continuous medication is desirable, it may be connected to an infusion bottle and fluid allowed to enter the catheter at the desired rate for as long a period as may be deemed necessary.

Before any real progress could be made with this method, it would be desirable to find out more about the tolerance of the tracheo-bronchial tree for solutions of all kinds, both as to amount and concentration.

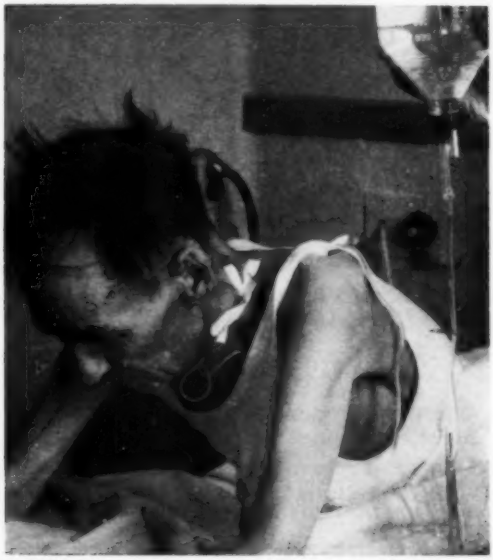

FIGURE 1: The catheter is shown in place on the patient's neck connected to an infusion bottle containing a solution of streptomycin.

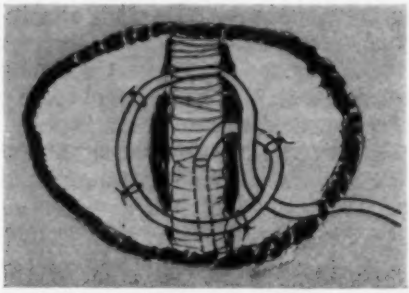

FIGURE 2: Diagram showing method of insertion of catheter into trachea. The dotted lines indicate the portion of the catheter in the tracheal lumen. The loose knot in the catheter and the sutures holding this to the strap muscles of the neck prevent the catheter from slipping out of the lumen with swallowing or with neck movements. The free end of the catheter is brought under the skin to issue on the back of the animal.

Since it would be advisable to test this on animals first, a procedure was drawn up for inserting a similar plastic catheter into the animal trachea. The purpose of this communication is to describe the method as we employ it at present in rabbits, and to suggest the possibilities of the method which we intend to explore. Later publications will deal with results.

The test animal we have selected has been the rabbit. This is a matter of convenience. Rats, mice and guinea pigs are too small, although the method could be used even in these animals. Dogs and cats could also be suitable subjects, but were not available with our present facilities.

The rabbit is first anesthetized with preliminary Nembutal intravenously and then carried on open ether anesthesia. An incision is made in the mid-line of the shaved neck and the trachea indentified.

A small opening is made with a scissors between two tracheal cartilages just below the thyroid gland. This opening is just large enough to allow the plastic catheter (No. 5 French) to be introduced. This is introduced

FIGURE 3: This shows the component parts of the bottle, metal plate and catheter assembly.

about an inch into the trachea until its tip is at a level estimated to be just above the carina. A loop is made in the catheter to be introduced.

The catheter with its loop now resting on the trachea is sutured to the strap muscle of the neck (Fig. 2). The loop is of some value in preventing the catheter from being pulled out of the trachea.

The free end of the catheter is then brought under the skin of the animal around to the back, through a stab wound prepared in the shaved back of the animal.

The net result is now a catheter protruding from the back of the animal which is in direct communication with the tracheo-bronchial tree. This could now be used, if necessary, to introduce materials intermittently—in any amounts and at any stated intervals desired.

In order to introduce fluids continuously it was necessary to devise an apparatus which could be sutured firmly to the back of the animal and which would carry a plastic bottle of sufficient size to allow infusion to proceed over several hours.

Several different types of apparatus have been devised. Fig. 3 shows a view of a metal plate which we have used for this purpose. This can be sutured to the animal and the bottle with its catheter attached to the catheter issuing from the animal.

Fig. 4 shows the assembly in place on the animal. Flow can be adjusted at several drops per minute. The rate is adjusted so as not to cause discomfort or coughing. In this way it is assured that the total dose is carried into the tracheo-bronchial tree.

There are numerous possibilities for further investigation with this method. Obviously, as intimated above, the tolerance of the tracheo-bronchial tree for solutions of all kinds can be tested.

Studies of infection of the tracheo-bronchial tree and lungs by introducing standard doses of the infectious agent, such as tubercle bacilli or staphylococci, should be easily accomplished. Studies of response of the infection to various antibiotics or other materials should follow as a natural consequence.

FIGURE 4: This shows the assembly in place and in operation in the intact animal.

The effect of irritants and carcinogens on the bronchial mucosa and the effect of temperature changes on the bronchial mucosa would also seem to be a fruitful source of study.

The carriage of particulate matter or radioactive solutions from the tracheo-bronchial tree and lungs to the draining lymph glands also suggests itself as a possibility for investigative study by this procedure.

SUMMARY

1. It is often desirable to introduce materials into the tracheo-bronchial tree in the course of investigation or treatment of bronchopulmonary diseases.

2. Aerosol methods do not provide sufficient fluid to the bronchi and operative procedures are cumbersome.

3. A method of introducing and leaving a cannula in the trachea of rabbits has been evolved and is described.

4. With this method, solutions can be introduced directly into the tracheo-bronchial tree in large amounts, either intermittently or continuously for days or weeks at a time.

5. It is proposed to use this method to try the effects of various solutions in the investigation and treatment of normal animals and others with induced diseases of the respiratory tract.

6. The use of this method in the treatment of human disease is suggested.

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My thanks are due to Dr. I. Hapner, Research Fellow in Pulmonary Diseases at the Jewish General Hospital in 1958-59, and to Messrs. Terence Seasman and Lionel Caplan, technicians in the Cardiac & Pulmonary Laboratories, for their aid in this work. Mr. Caplan was especially helpful in originating and designing the backplate and bottle on the animals to connect with the catheter.

RESUMEN

1. A veces es de desearse el introducir materiales dentro del árbol traqueobrónquico al investigar las enfermedades broncopulmonares.

2. Los aerosoles no proporcionan suficiente líquido dentro de los bronquios y los procedimientos son molestos.

3. Se ha descrito un método consistente en introducir una cánula en la tráquea de conejos.

4. Por este medio se pueden introducir grandes cantidades de soluciones directamente en el árbol traqueobrónquico ya sea intermitente o contínuamente por días o semanas.

5. Se propone que se use este método para ensayar varias soluciones en la investigación y tratamiento de animales normales y en animales con enfermedades provocadas en las vías respiratorias.

6. Se segiere la aplicación de este método en el hombre enfermo.

RESUMÉ

1. Il est souvent désirable d'introduire des produits dans l'arbre trachëo-bronchique au cours d'investigations ou de traitement des affections broncho-pulmonaires.

2. Les aérosols n'apportent pas un liquide suffisant aux bronches et les procédés opératoires sont gênants.

3. Une méthode d'introduction et de retrait d'une canule dans la trachée de lapins a été mise au point et est décrite.

4. Avec cette méthode, des solutions peuvent être introduites directement dans l'arbre trachéo-bronchique en grandes quantités soit de façon intermittente, soit de façon continue, pour des jours ou des semaines en une fois.

5. Les auteurs proposent d'utiliser cette méthoed pour essayer les effets de différentes solutions pour l'examen et la traitement d'animaux normaux et d'autres porteurs d'effections provoquées de l'arbre respiratoire.

6. L'utilisation de cette méthode dans le traitement des affections humaines est évoquée.

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Observations on the Cytology of Tracheobronchial Secretions Collected by a New Technique*

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Expectorated sputum and secretions aspirated through the bronchoscope frequently contain contaminants from the oral cavity.[1] Because of this, it has not been possible often to accurately determine the cytology of the tracheobronchial tree in normal or diseased patients. Carabelli[2] after studying secretions obtained by bronchoscopic aspiration reported that they normally contained ciliated columnar, goblet, undifferentiated bronchial, and squamous cells in addition to macrophages. He attributed blood elements to trauma, and did not consider them a part of the normal cytogram. Moreover, he believed that squamous cells probably represented elements which had been aspirated deep into the bronchial tree, presumably from the supraglottic region. For this reason little importance was attributed to their presence. In suppurative bronchopneumonitis he found leukocytes prominent and predominating. It has been our experience[1] that under both normal and pathological conditions, in the absence of instrumentation, oral contents are rarely aspirated into the tracheobronchial tree.

Recently a procedure was developed, which permitted us to obtain secretions by transtracheal aspiration.[3] This enabled us to study not only the bacteriology, but also the cytology of the specimens.

Prior to the procedure the patient is given a suitable dose of barbiturate for sedation. A small pillow is placed beneath the patient's shoulders and the neck is extended as he lies supine. The anterior neck is prepared with an antiseptic and draped with sterile towels. A small cutaneous wheal of 0.5 per cent procaine is then made over the membranous trachea about 1 centimeter below the lower border of the cricoid cartilage in the midline. Following this a 15 gauge needle is inserted through the wheal into the trachea the point being directed caudad. A 6" length of sterile polyethylene tube (internal diameter 0.034 inch and outside diameter 0.05 inch) is inserted into the trachea through the needle, after which the latter is withdrawn. Following this about 1 to 3 cc. of 0.9 per cent saline solution is injected into the tube with the aid of a syringe fitted with a 20 gauge needle. The patient is encouraged to cough as suction is applied to the tube by the same syringe. If an adequate specimen of sputum is obtained mucous will be noted in the syringe. If bleeding should occur through the puncture site it can easily be controlled by pressure with a sterile gauze pledget.

Immediately after the secretions are aspirated by tracheal puncture a large drop is placed upon a glass microscope slide previously coated with a thin layer of Mayer's albumin. A smear is made by apposing the surface to that of another slide and pulling the slides apart in a plane parallel with their surfaces. The slides are immediately fixed in ether and alcohol according to the Papanicolaou[4] method (No. 268). One slide

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is then stained in accordance with the Papanicolaou technique; the other is allowed to dry, after which it is stained by standard Gram's method. The specimens are carefully examined under the microscope to identify bacteria and cell types, and to estimate relative numbers of the various types of cells. The tracheobronchial secretions are also routinely cultured for pyogenic organisms as previously described.[1]

Results

In Table 1 cases are listed according to diagnosis. There was no "normal" subject. All had pulmonary infiltration by roentgenogram. All had polymorphonuclear leukocytes in the tracheobronchial aspirates. However, the proportion of patients with "many" polymorphonuclear leukocytes is higher in the group which had carcinoma or evidence of active pulmonary infection (including infectious tuberculosis, blastomycosis, pulmonary abscess, and aspergillosis) than in the group which did not (including noninfectious pulmonary tuberculosis, undiagnosed pulmonary disease or subphrenic abscess).

The role of inhaled irritants in the production of purulent sputum may be surmised from the fact that 52 per cent of the patients smoked over 20 cigarettes a day and only 17 per cent did not smoke. Only 5 per cent gave a history of exposure to dusts which might have been responsible for pneumoconiosis. Twenty seven per cent had recently lived in large cities, where air pollution might have provided a source of irritation to the lower respiratory tract. It is interesting to note that of the 82 patients included in this study there were only seven who gave a history of smoking less than 20 cigarettes a day and offered no evidence of active pulmonary suppuration. It is obviously difficult to locate subjects who are not exposed to some type of bronchial irritant, either chemical or bacterial.

TABLE 1 — CYTOLOGY OF TRACHEOBRONCHIAL SECRETIONS ACCORDING TO DIAGNOSIS

Diagnosis	Number of Cases	Cytogram: Polys. M	Polys. F	Polys. O	Monos. M	Monos. F	Monos. O	Squam.	Colum.	Macro.	Atypia	Malig.
Infectious Tuberculosis	45	38	7		22	23		26	42	28	5	
Noninfectious Tuberculosis	11	5	6		4	7		5	11	3		
Blastomycosis	1		1			1		1	1			
Aspergillosis with Silicosis	1	1				1		1	1		1	
Pulmonary Abscess	2	2			2			1	1	1	1	
Subphrenic Abscess	1		1			1		1	1			
Undiagnosed Pulmonary Disease	14	8	6		5	8	1	9	13	7	3	
Bronchogenic Carcinoma	7*	5	2		4	3		6	7	3	2	4
Total	82											

Polys. = Polymorphonuclear leukocytes; Monos. = Monocytes and lymphocytes; Squam. = Squamous epithelial cells; Colum. = Columnar epithelial cells; Macro. = Macrophages; Malig. = Malignant cells; Micro. = Microscopic. M = Many; F = Few; O = None.

*One of these patients also had noninfectious tuberculosis.

In translating microscopic findings to macroscopic terminology it should be noted that although 20 of our patients yielded expectorations which were described by the examiner as mucoid, every patient had polymorphonuclear leukocytes in the tracheal aspirates.

Reference to Table 1 reveals that it was possible to diagnose or suspect carcinoma in a single specimen in a considerable proportion of the patients who had the disease. The high incidence of squamous cells, which were of the "superficial" type is interesting, since these do not normally originate in the lower respiratory tract unless metaplasia exists.[5] It appears unlikely that many of these cells could have originated above the vocal cords since accumulated bacteriological data indicate that contamination of lower respiratory tract with pharyngeal contents is rare.[1] Since metaplasia of the bronchial mucosa may occur as the result of irritation, it is noteworthy that 51 per cent of the patients in this series were found to have reddened bronchial mucosa at bronchoscopy. The consistent presence of columnar bronchial epithelium is considered an indication of the adequacy of the specimen.

Discussion

From the evidence presented in this study it appears likely that polymorphonuclear leukocytes are to be found in the respiratory tract under a variety of conditions. Where expectorations are purulent leukocytes tend to be more abundant. However, they are frequently present when the sputum is grossly nonpurulent. In the presence of infection, carcinoma, or following inhalation of irritants this is understandable. However, there is some evidence that leukocytes frequently occur in the secretions of normal individuals. Such conclusions are based upon the knowledge that the lungs normally tend to filter white cells from the circulation.[6] The difference between the normal and abnormal with respect to the number of leukocytes in the tracheobronchial secretions may be only relative.

With the technique employed in this study it appears that contaminants from the supraglottic region are rare. Since squamous epithelial cells are not normally found below the vocal cords, it is probable that their presence, in tracheal aspirates obtained by the method described, may indicate the presence of metaplasia of the bronchial epithelium.

SUMMARY

Tracheal secretions were collected from 82 patients with various pulmonary diseases by a method which permitted little contamination. Secretions from those with both infectious and noninfectious tuberculosis, blastomycosis, aspergillosis and silicosis, subphrenic abscess, pulmonary abscess, carcinoma, and various undiagnosed pulmonary conditions contained polymorphonuclear leukocytes. Grossly nonpurulent sputum always contained leukocytes, although usually not as many as purulent sputum. Squamous cells were found in a considerable number of samples, and under the conditions of this study are considered evidence of metaplasia of the bronchial mucosa. Evidence of bronchial irritation was found in 51 per cent of the patients by bronchoscopy. Only three in this series yielded no evidence of infection, carcinoma or inhalation of irritants. It appears likely that leukocytes may be found in a large proportion of tracheobronchial secretions from normal individuals.

RESUMEN

Se han colectado las secreciones traqueales de 82 enfermos con varios padecimientos pulmonares por un método que permite poca contaminación.

Fueron secreciones provinientes de tuberculosis infectante y no infectante, blastomicosis, aspergilosis y silicosis, absceso subfrénico, absceso pulmonar, carcinoma y varios padecimientos no diagnosticados.

Los esputos aparentemente no purulentos siempre contenían leucocitos, aunque habitualmente no tantos como los purulentos.

Se encontraron celdillas escamosas en un número considerable de muestras y bajo las condiciones de este estudio, se consideran como evidencia de metaplasia de la mucosa bronquial.

Prueba de irritación bronquial se encontró en 51 por ciento de los enfermos, por broncoscopía. Sólo tres de esta serie no dieron evidencia de infección, carcinoma o inhalación de irritantes. Parece posible que los leucocitos pueden encontrarse en gran proporción de secreciones de individuos normales.

RESUMÉ

Des sécrétions trachéales furent recueillies sur 82 malades atteints de différentes affections pulmonaires, par une méthode qui permit peu de contamination. Les sécrétions de ceux atteints de tuberculose aussi bien bacillifére que non bacillifére, de blastomycose, d'aspergillose et de silicose, d'abcès sous-phrénique, d'abcès pulmonaire, de cancer et de différents états pulmonaires de diagnostic indéterminé contenaient des leucocytes polymorpho-nucléaires. Une expectoration macroscopiquement non purulente contenait toujours des leucocytes, bien qu'habituellement en quantité moindre que l'expectoration purulente. Des cellules épidermoides furent trouvées dans un nombre considérable d'échantillons, et dans les conditions de cette étude, sont considérées comme la preuve métaplasie de la muqueuse bronchique. La preuve d'ne irritation bronchique fut trouvée dans 51% des malades par la bronchoscopie. Trois malades seulement sur ce groupe ne fournirent aucune preuve d'infection, de cancer ou d'inhalation de substances irritantes. Il parait vraisemblable que des leucocytes peuvent être trouvés dans une grande proportion des sécrétions trachéobronchiques chez les individus normaux.

ZUSAMMENFASSUNG

Es wurde Luftröhrenschleim von 82 Kranken mit den verschiedensten Lungenkrankheiten gesammelt und zwar mit einer Methode, die nur eine geringe Verunreinigung zuläßt. Sekret sowohl von infektiöser, als auch nicht infektiöser Tuberkulose, von Blastomykose, Aspergillose und Silikose subphrenischem Abszess, Lungenabszess, Lungencarzinom und verschiedenen nicht diagnostizierten Lungenerkrankungen enthielt polymorphkernige Leukozyten. Nach dem makroskopischen Aussehen enthielt nicht eitriges Sputum immer Leukozyten, wenngleich gewöhnlich nicht so reichlich wie eitriges Sputum. Epithelzellen fand man in einer beträchtlichen Zahl von Sekretproben, und unter den Bedingungen dieser Untersuchung werden sie als Beweis für eine Metaplasie der Bronchialschleimhaut gewertet. Beweise für einen Reizzustand der Bronchien fanden wir bronchoskopisch bei 51% der Kranken. Nur dreimal ergab sich in unserem Material kein Anhalt für eine Infektion, Carzinom oder Inhalation von Reizstoffen. Es dürfte sehr wahrscheinlich sein, daß man die Leukozyten in einer Vielzahl von Tracheobronchial-Sekreten bei normalen Menschen finden kann.

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BRONCHOESOPHAGEAL FISTULA IN A CASE OF TUBERCULOSIS CURED BY CHEMOTHERAPY

A case is described in which bronchoesophageal fistula arose from a tuberculous lymph node in the mediastinum. Gastrostomy was performed, and the patient was treated with streptomycin and INH; this resulted in healing of the fistula. One year later, the patient was examined. Her general condition was good and there was no evidence of the fistula. The result of treatment should be considered permanent.

Trzcinski, K.: "Bronchoesophageal Fistula in a Case of Tuberculosis Cured by Chemotherapy," *Gruzlica*, 28:819, 1960.

EFFECT OF HISTAMINE AND SEROTONIN ON BRONCHIAL MUSCLES OF GUINEA PIGS

A comparative evaluation is made on the bronchospastic action of serotonin and histamine on isolated trachea and with aerosol treatment of guinea pigs.

Equal doses of the two amines used, exerted analogous effects as to intensity and duration in both experimental techniques.

An additive effect is noted when the two substances are used together. The combined effects of the two amines most probably are responsible for the anaphylactic bronchospastic shock in guinea pigs.

Mariani, L.: "Effect of Histamine and Serotonin on Bronchial Muscles of Guinea Pigs," *Atti, Accad. Med. Lombarda*, 15:165, 1960.

Preoperative Diagnosis of Tuberculous Endobronchitis

A Radiologic Study

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Since the advent of antimicrobial agents, the incidence of tuberculous lesions in the bronchi as seen bronchoscopically has markedly decreased. The lack of epithelial lesions, however, does not negate the possibility of there being active submucosal tubercles. Thomson and Kent (1958) reported 44 cases with proved histological evidence of endobronchial disease. In all these 44 cases, preoperative bronchoscopy was essentially normal; however, 10 of the patients had shown some evidence of endobronchial disease on previous examination. It is not surprising that normal bronchoscopy findings are reported in spite of an active endobronchial disease. This is because with antimicrobial therapy, there is rapid healing or repair of an epithelial surface. Hardy and Samson (1956) described a quiescent bronchus which was essentially negative on endoscopic examination, but was not a healed bronchus in a histopathologic sense. In a review of 602 resected specimens, Olson and coworkers (1953) reported that 37.9 per cent of patients with normal bronchoscopic findings showed evidence of endcbronchial disease on microscopic examination of the resected specimen.

FIGURE 1

FIGURE 2

FIGURE 1: Bronchotomogram for cavitary disease, apicoposterior segment, left upper lobe. Note the irregularity of apicoposterior segmental bronchus with failure to fill the cavity. It was interpreted as due to tuberculous endobronchitis. An apicoposterior segmental resection was done and active endobronchial disease confirmed histologically.

FIGURE 2: Bronchotomogram for residual necrotic disease, posterior segment, right upper lobe. Note the narrowing of right upper lobe bronchus and corrugations in the floor due to tuberculous endobronchitis. This necessitated right upper lobectomy. Active tuberculous endobronchitis proved histologically.

What is the significance of these occult tuberculous lesions in the bronchi? Perhaps the most dreaded complication of tuberculous endobronchitis is the post-operative development of a bronchopleural fistula due to the breaking down of the stump. If viable tubercle bacilli remain within a cavity despite long-term chemotherapy, then the bronchial tree is constantly contaminated and the possibility is great that tuberculous endobronchitis will ensue and persist and such cases will continue to pose a problem for the clinician.

It is my impression that preoperative diagnosis of endobronchial tuberculosis can be made in a large number of cases by bronchography and bronchotomography in spite of negative bronchoscopic findings. Bronchograms have been done by trans-glottic method using Dionosil as contrast media. Immediately a straight skiagram and tomogram (laminogram) at the most representative level is taken. The following x-ray films are illustrated.

In this country with high incidence of tuberculosis, many patients have had inadequate and irregular chemotherapy prior to hospitalization and harbor drug-resistant tubercle bacilli. Proper evaluation of the existence of tuberculous endobronchitis is essential to decide the extent and type of surgery in these cases. In 33 bronchoscopically negative and histologically proved cases of tuberculous endobronchitis, preoperative diagnosis of tuberculous endobronchitis could be made in 24 (75 per cent) cases by bronchotomography. The chief radiological criteria of tuberculous endobronchitis are: 1) irregularity and corrugations of bronchial

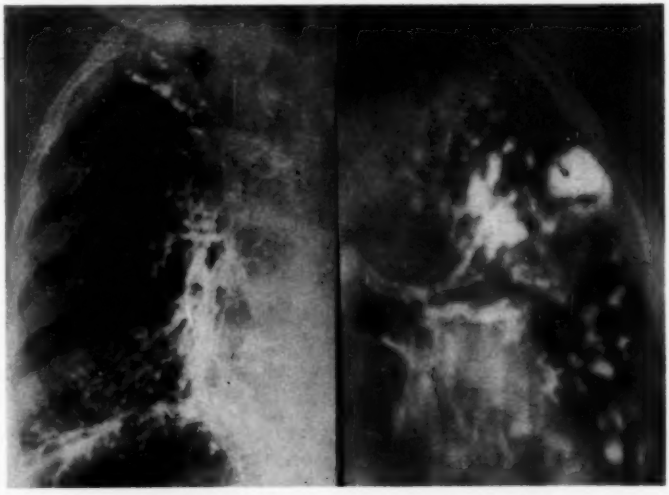

FIGURE 3 FIGURE 4

FIGURE 3: Bronchogram for atelectatic right upper lobe. Note the clear outline of right upper lobe bronchus, interpreted as absence of endobronchial disease. Right upper lobectomy was done and no evidence of tuberculous endobronchitis detected histologically. FIGURE 4: Bronchotomogram reveals bronchiectasis and extensive cavitary disease left uuper lobe. Note the marked irregularity of the left upper lobe bronchial mucosa. Left upper lobectomy was done and histopathology revealed extensive endobronchial tuberculosis.

mucosa as compared with the smooth outline of adjoining healthy segments; 2) narrowing and failure to fill the involved segments when the surrounding healthy segments are properly filled.

SUMMARY

Preoperative diagnosis of tuberculous endobronchitis is important to decide the type and extent of surgery in the treatment of pulmonary tuberculosis. In a great majority of cases it can be assessed by preoperative bronchotomography in spite of negative bronchoscopic findings.

RESUMEN

El diagnóstico preoperativo de la traquebronquitis tuberculosa es importante para decidir el tipo y la extensión de la cirugía en tratamiento de la tubercuulosis pulmonar.

En la gran mayoría de los casos puede apreciarse por la broncotomografía a pesar de los hallazgos broncoscópicos negativos.

RESUMÉ

Le diagnostic préoperatoire d'atteinte tuberculeuse de l'endobronche est important pour décider du type et de l'étendue de la chirurgie dans le traitement de la tuberculose pulmonaire. Dans la grande majorité des cas, il peut être établi par la bronchotomographie préoperatoire alors que les constatations bronchoscopiques restent négatives.

ZUSAMMENFASSUNG

Die Erkennung der tuberkulösen Endobronchitis vor einer Operation ist wessentlich für die Entscheidung von Art und Ausdehnung des Eingriffes bei der Behandlung der Lungentuberkulose. In einer beträchtlichen Vielzahl von Fällen lassen sich trotz negativer bronchoskopischer Befunde — mittels einer vor der Operation vorgenommenen Schichtaufnahme der Bronchien-die richtigen Schlüsses ziehen.

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ANOMALOUS PULMONARY VENOUS DRAINAGE

The authors studied 43 proved cases of anomalous pulmonary venous drainage. This condition is practically always associated with other malformations, especially atrial septal defect. The veins usually connect with the right atrium, but they may connect with the venae cavae, the azygos, the portal vein, the coronary sinus, etc.

This condition was arbitrarily separated into four types: 1) the condition is isolated; 2) it is associated with atrial septal defect; 3) all of the pulmonary venous system connects with the right half of the heart; 4) it coexists with more serious malformations of the heart such as tetralogy of Fallot, pulmonary stenosis, etc.

The study which affords the best diagnostic features in any type is catheterization of the heart, and especially the test consisting of the temporary occlusion of one or several veins, that is, the identification of the veins connecting abnormally and its eventual occlusion with the Dotter-Lukas catheter.

The occlusion of one of the veins and the persistence of a left-to-right shunt permits one to suspect that: a) there is more than one vein connecting abnormally; b) there is an associated atrial septal defect. The temporary occlusion of the right branch of the pulmonary artery interrupts the arteriovenous shunt at the atrial level if there were an atrial septal defect.

Canale, M., Espino Vela, J., Rubio, V., Ruiz, M. C., and Uzun-Haendel, T.: "Anomalous Pulmonary Venous Drainage," *Arch. Inst. Cardiol. de Mexico*, XXX:583, 1960.

ACCLIMATIZATION TO CARBON DIOXIDE

Although a sufficiently steady state for respiratory response measurements is reached within 5 to 10 minutes, the tissues probably to dot come into complete equilibrium for days. The renal compensations are also very slow, as noted above. For this reason, it is unsafe to presume that changes occurring in the first few hours of hypocapnia or hypercapnia resemble the long-term adjustments of true acclimatization. Transient alterations in respiratory exchange ratio, urinary composition, and arterial pH, for example, do not persist into the truly acclimatized state.

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Experimental Lung Resection During Vascular and Bronchial Occlusion*

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Introduction

Lung resections of less than a lobe are apt to be bloody and thus imprecise when the dissection is carried outside the segmental plane. If clamps are used to avoid a bloody and bubbling field and a wedge type of resection is done, some normal tissue is resected and other normal tissue is impaired by the repair. Better techniques are needed. This need is particularly apparent if multiple small lesions must be resected or if the disease passes over segmental planes. A lung with multiple emphysematous blebs, for example, presents these problems.

The technique we have employed to facilitate subsegmental lung resections is a simple one in which the lung is made bloodless and essentially airless prior to resection by clamping the blood vessels and the bronchus at the lung root.

Method

Thoracotomy was performed in 25 adult mongrel dogs through the right third or left fourth intercostal space. Anesthesia was obtained with pentobarbital sodium and morphine sulfate. Breathing was maintained with an automatic respirator.

The animals were divided into three groups. Group I consisted of six dogs in which the result of clamping the pulmonary arteries, veins and the bronchus in various combinations and sequences was observed in order that we might learn a satisfactory way to produce ischemia. In some dogs, the whole pedicle was clamped with one clamp, while in others, the pulmonary artery, the vein and the bronchus were clamped individually and in varying order.

In group II, consisting of 8 animals, the pedicle of the right upper lobe was freed and the pulmonary artery clamped with an occluding tape. Immediately afterwards, the inspiratory pressure produced by the respirator was raised from the usual level of 16 millimeters of mercury to 25-30 millimeters of mercury. This increased inspiratory pressure was applied intermittently for from 3 to 15 minutes, and finally, at the height of a forced inspiration, the pulmonary vein or veins and the corresponding bronchus were occluded with tape tourniquets.

Group III consisted of 11 animals in which the procedure of group II was repeated except that the whole left lung was made ischemic. After the components of the pedicle were clamped, wedge resections were done to make certain that the lung was ischemic. The defects of the lung thus

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produced were sewed up. After a period of complete ischemia ranging from 60 to 90 minutes the pulmonary veins, the bronchus and the pulmonary artery were unclamped in that order.

Tape was used to occlude the vessels of the right upper lobe. However, the bronchus was clamped with either a straight Potts patent ductus clamp or a Satinsky vena cava clamp. These clamps were also frequently used to clamp the hilar vessels in order that one might determine whether or not this method was adequate for the experiment and inocuous to the clamped vessel.

The dogs in group II were sacrificed by exsanguination on 10 to 40 days and in group III in 42 to 66 days after the experiment.

Patency of the bronchial system of vessels was checked by x-ray films as follows: the thoracic portion of the aorta was ligated just beyond the arch and at the level of the diaphragm. About 100 milliliters of barium sulfate suspension were injected into the thoracic aorta, and then the heart, lungs and aorta were removed *en masse* from the chest cavity and x-rayed. The filled bronchial arteries were evident. The integrity of the previously clamped bronchi was checked by x-ray film taken after the main divisions of the main stem bronchi had been tied and both trachea and main stem bronchi filled with hypaque solution. Finally, the main vessels and bronchi were inspected and photographs taken.

As a further test of the functional capacity of the left lung which had been rendered ischemic, right pneumonectomy was done in four dogs of group II in 18, 19, 34, and 42 days after the ischemia experiment.

Results

Survival was not observed in group I, since the purpose was to perfect the technique of ischemia. Studies in this group suggested that the

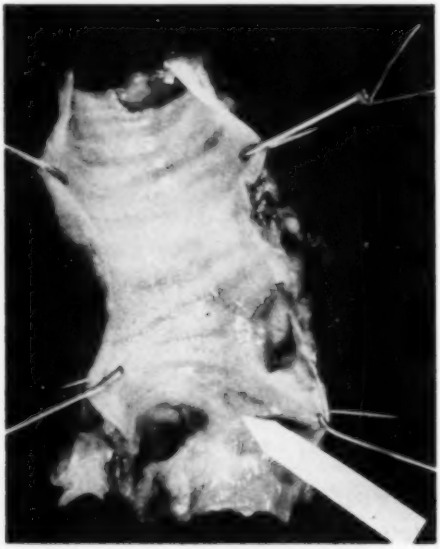

FIGURE 1: The bronchus appears undamaged at the site of clamping which is indicated by the arrow.

optimal time for emptying the lung of the residual blood after the pulmonary artery had been clamped and before the corresponding veins and bronchus were occluded was at least five minutes.

All of the 19 animals of group II and group III had long healthy survival, except one animal of group III which died six days postoperatively of bleeding resulting from faulty suturing of the wedge resections done during surgery.

Patency of the previously clamped bronchi (Fig. 1, 2) and main vessels was the rule without exception. The bronchial vessels were also patent as seen in the x-ray films and in histologic sections (Fig. 3).

All four dogs subjected to pneumonectomy survived the operation, but one died 72 hours after surgery. Autopsy showed a heavy red, wet left lung and intussusception of about three fourths of the stomach into the esophagus presumably because of the abnormally decreased intrathoracic pressure resulting from the pneumonectomy. The left lung, also, had extensive dense adhesions to the chest wall as a result of the ischemic opration. The other three dogs were alive and well 9, 21 and 22 days after the removal of the right lung and 27, 59 and 64 days after the left lung had been rendered temporarily ischemic. Autopsy showed normal lung tissue and patent bronchi and main vessels.

Comment

Attempts at reducing the bleeding during resection of portions of lung tissue are recorded as early as 1876 when Kirchhoff[4] exteriorized a diseased lobe, applied a tourniquet around the secondary hilus and excised the lobe. Since then, instruments[4,5] as well as methods[9] have been devised to render dissection through the lung tissue as bloodless as possible.

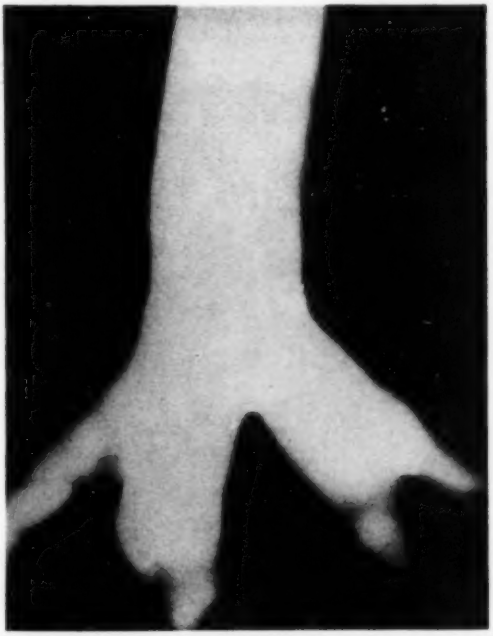

FIGURE 2: X-ray film showing that the left main stem bronchus has normal lumen without stricture many weeks after clamping (see text).

Some of the instruments as that of Garre and Quincke (1912)[4] and of Shenstone (1937)[6] were applied to the whole hilus, whereas that of Schumacher (1912)[4] was applied upon any part of the lung distal to which pulmonary excision was to be performed.

An ingenious method of excising a diseased lobe was used by Sauerbruch and Schumacher (1911).[5] They ligated the corresponding pulmonary artery one to two weeks before excising a bronchiectatic lobe. This method was supposed to shrink the diseased lobe and render it almost entirely bloodless.

Modern thoracic surgeons have utilized temporary hilar ligation of the bronchus and the vessels in the excision of pulmonary arteriovenous fistulas[2,7] and have studied the effects of occlusion of the hilus on the survival and pulmonary function of dogs.[2] Whereas these surgeons have reported success in obtaining relatively avascular fields, Blades and his associates have repeatedly[1,2] emphasized the fact that "in addition to individually ligating the main hilar vessels and the bronchus, further insurance of total ischemia was . . . effected by placing a crushing tourniquet . . . around the entire pulmonary hilus."[1] It is our opinion that even then one cannot get a completely bloodless field which one gets, for instance, in hand surgery done with the help of an arm tourniquet. Each lung holds about 100 to 300 cc. of blood trapped in its vastly numerous capillaries, the total inner surface of both lungs being close to 700,000 square centimeters.[3] In addition, in our group I experiments even after complete severance of the lung pedicle there was so much bleeding from wedge resections that one might think there was a hidden source of blood within that lung producing several milliliters of blood each minute.

Success was obtained in our efforts to produce a bloodless lung resection when we not only clamped all hilar structures tight, but also added the step of emptying the lung or lobe of the residual blood. As described under Methods, the pulmonary artery was first occluded and the inspiratory pressure was then increased to 25 to 35 millimeters of mercury and intermittently kept at this high level for at least 5 minutes. Only then would a cut into lung tissue resemble a cut through a moist sponge. This principle proved to be correct in the human cases in which it has been employed, and use of this technique especially in cases with emphysematous blebs will be reported later. Moreover, according to the present work, in the humans as well as in the dogs, clamping the pulmonary veins was necessary in spite of the low pressure within these vessels.

Another interesting fact was discovered in our studies. The lung tissue, canine or human, can withstand total ischemia for at least 60 minutes and probably more. Bosher[2] doubted the statistical significance of the figures and the validity of conclusions of Blades[1] that 30 minutes is the upper limit of safe vascular occlusion of the lung. We are inclined to agree with Bosher. Moreover, we have proved to our satisfaction that doubling that period, *i.e.* making it 60 minutes, would not be injurious to either canine or human lung tissue.

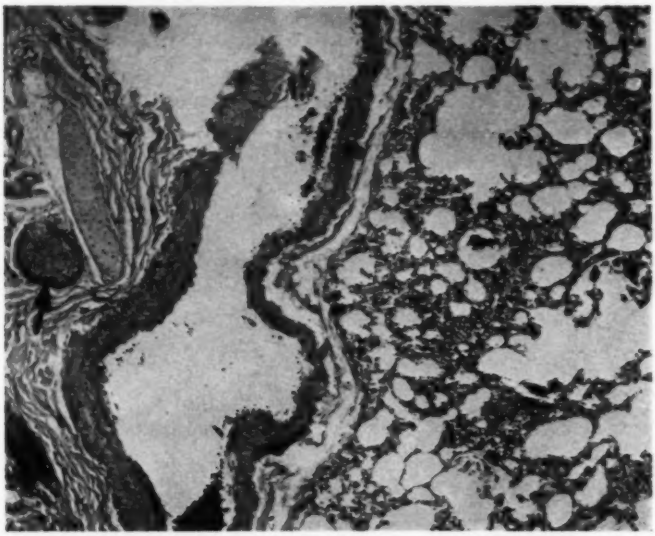

FIGURE 3: Normal histologic appearance of lung parenchyma and bronchus in one lung rendered ischemic during life. Arrow points to the barium filled bronchial vessel which was obviously patent long after the ischemia experiment.

SUMMARY

A perfected technique of producing temporary, complete pulmonary ischemia is described. Any sublobar lung resection can be done in a bloodless field by this technique. Its safety has been proved by experimentation in 19 dogs in which either a lobe or an entire lung were rendered ischemic for 60 minutes.

RESUMEN

Se discribe un técnica para producir isquemia pulmonar completa y temporal. Cualquiera resección sublobar puede hacerse en campo exangüe con esta técnica. Su seguuridad ha sido probada en 19 perros en experiencia y de los que se ha logrado que ya sea un lóbulo o un pulmón entero permanezcan isquémicos por 60 minutos.

RESUMÉ

Un procédé perfectionné de production temporaire d'ischémie pulmonaire complète est décrit. Toute résection sublobaire du poumon peut être faite dans un champ exsangue par cette technique. Son absence de danger a été mise en évidence par l'expérimentation chez 19 chiens, chez lesquels soit un lobe soit le poumon entiers étaient rendus ischémiques pendant 60 minutes.

ZUSAMMENFASSUNG

Beschreibung einer fehlerlosen Technik zur Herstellung einer temporären kompletten pulmonalen Ischämie. Mit dieser Technik kann jede sublobuläre Lungenresektion in einer blutleeren Gebiet ausgeführt werden. Ihre Sicherheit wurde erwiesen durch Experimente an 19 Hunden, bi denen entweder ein Lappen oder eine ganze Lunge während 16 Minuten blutleer blieb.

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PATHOLOGIC AND ANATOMIC CHANGES IN BRONCHIAL STUMPS AT DIFFERENT PERIODS FOLLOWING PNEUMONECTOMY AND LOBECTOMY

Histologic study of 30 bronchial stumps in patients who died at various periods of time after lung resection and lobectomy for different diseases has shown that in uncomplicated cases, the formation of the epithelized connective tissue adhesion takes place to the twelfth day postoperatively. The formation of the adhesion was markedly retarded by the development of suppurative processes in pleural cavities that complicated the postoperative course of the disease. The formation of the fibrous adhesion commenced from distal stump portions. Reactive inflammatory changes about tantalum clips were considerably less than that about silk ligatures, the very region where not infrequently suppurative processes start the development. Of 11 bronchial cysts studied in 7 cases, the cause of the origin was suppuration at suture site and ligature disruption; in 2, destruction of the stump tissue involved by cancer and associated with suppurative processes. In one patient in the region of the stump, the mycotic process has developed. In another patient, there was inadequate stump suture.

Lazarev, V. I.: "Pathologic and Anatomic Changes in Bronchial Stumps at Different Periods Following Pneumonectomy and Lobectomy," *Chest Surgery (USSR)*, 85:59, 1960.

Tracheal Fenestration: A Critical Evaluation*

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Surgery often serves as a valuable adjunct in the management of chronic obstructive pulmonary emphysema. The operation of tracheostomy contributes to treatment by: (1) reducing the respiratory dead space, (2) diminishing the collapse of the bronchi on expiration, (3) facilitating tracheobronchial aspiration, and (4) permitting the local instillation of therapeutic agents. Recently modifications have been advocated to substitute for the standard tracheostomy because of certain alleged inconveniences of the latter procedure. One such proposal has been to create a tracheal fenestration[1,2] which would lend itself to intermittent aspiration and local therapy, but remain closed at other times.

The purpose of this report is to compare tracheal fenestration and standard tracheostomy in the treatment of chronic obstructive pulmonary emphysema by evaluation of the clinical course and the determination of blood gas studies in a series of patients.

Case Material

All patients with severe chronic obstructive pulmonary emphysema admitted to the Coral Gables Veterans Administration Hospital with an initial arterial carbon dioxide tension of 55 mm. of Hg. or more and having either a tracheostomy or a tracheal fenestration were studied. The period covered was between January 1, 1958 and January 1, 1960.

Eighteen patients had adequate serial blood gas determinations, and these constitute the basis of this report.

Between April and October, 1959 a tracheal fenestration as described by Rockey[1,2] was performed on all patients with chronic obstructive pulmonary emphysema who required a tracheal surgical procedure and whose condition permitted transport to the operating room. Ten fenestration operations were performed during this time interval, and adequate data is available in seven of these. During this same period, 11 patients with standard tracheostomies had adequate studies.

Results

Arterial carbon dioxide tensions and oxygen saturations were measured in the surgically treated patients. The most abnormal pre-treatment values were arbitrarily selected to be compared with the best post operative determinations. The results are shown in Tables 1 and 2. Patient survival following the performance of a tracheal operation is represented in Table 3.

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TABLE 1—ARTERIAL CARBON DIOXIDE REDUCTION AFTER TREATMENT

Standard Tracheostomy	Tracheal Fenestration
1. 0 mm. Hg.	1. 0 mm. Hg.
2. — 3	2. — 3
3. — 5	3. — 6
4. —10	4. —10
5. —12	5. —12
6. —15	6. —14
7. —15	7. —19
8. —16	
9. —18	

Difference was obtained by subtracting the lowest post treatment figure during the first three weeks of therapy from the highest pre treatment figure.

TABLE 2—ARTERIAL OXYGEN SATURATION AFTER TREATMENT

Standard Tracheostomy	Tracheal Fenestration
1. +1 per cent	1. 0 per cent
2. +4	2. +9
3. +8	3. +22
4. +19	4. +28
5. +22	5. +35
6. +22	6. +37
7. +38	7. +46
8. +40	

Values obtained represent the difference between the lowest pre treatment oxygen saturation and the highest post treatment figure.

TABLE 3—SURVIVAL AFTER TRACHEAL OPERATION

Standard Tracheostomy	Tracheal Fenestration
1. Dead 1 day	1. Dead 7 days
2. Dead 5 days	2. Dead 11 days
3. Dead 6 days	3. Dead 21 days
4. Dead 10 days	4. Dead 4 months
5. Dead 11 days	5. Dead 8 months
6. Dead 47 days	6. Dead 8 months
7. Dead 6 months*	7. Alive 13 months
8. Dead 6 months*	
9. Alive 7 months*	
10. Alive 12 months*	
11. Alive 24 months	

*Tracheostomy Permanent

Discussion

Chronic obstructive pulmonary emphysema is a potentially lethal disease for which a single simple therapeutic measure is not yet available. Diligent and aggressive medical therapy is still the only effective method of palliating this entity. The surgical creation of a tracheal stoma plays only a limited role in the overall therapeutic scheme. The eventual result is related more to the vigor of proper medical management than to the fact that a tracheal opening is present. However, there is no question that a tracheal stoma is of great aid when it can serve the functions enumerated in the introduction, namely: (1) reducing the respiratory dead space, (2) diminishing the collapse of the bronchi on expiration, (3) facilitating tracheobronchial aspiration, and (4) permitting local instillation of therapeutic agents.

In the patients reported, there was essentially no difference in results when tracheal fenestration was compared to standard tracheostomy (Tables 1, 2 and 3). Therefore, the merit of tracheal fenestration must be assessed on criteria other than improvement in blood gas determinations and prolongation of life—because within these parameters there is no difference.

The only possible advantage of the tracheal fenestration operation, then, is that the patient may phonate normally and that a tracheostomy tube is not worn. Since a permanent standard tracheostomy tube may be corked, there remains finally only the advantage of not wearing the tracheostomy tube. That the wearing of such a tube is a factor of minor importance is attested by a number of our patients who have worn permanent standard tracheostomy tubes without difficulty.

Having first considered the possible advantages of tracheal fenestration, it is appropriate next to examine certain of its potential disadvantages. It is apparent that a tracheal fenestration can only serve two of the functions of a tracheal stoma: facilitating tracheobronchial aspiration and permitting the local instillation of therapeutic agents. Because the fenestration is closed when not intubated, it cannot fulfill the other purposes of a tracheal stoma, reducing the respiratory dead space and diminishing the collapse of the bronchi on expiration. Tracheal fenestration, therefore, can at best serve only a part of the functions of a tracheostomy. This fact was demonstrated by the necessity to introduce a small tracheostomy tube through the fenestration in several patients when readmitted for exacerbation of their disease.

SUMMARY

1. Surgery plays an important but limited role in the treatment of the emphysema patient. By far, the intensity of application of medical measures is the dominant factor in determining the final outcome.

2. Tracheal fenestration yields results which in our hands are no better than standard tracheostomy. When examined critically, tracheal fenestration has the disadvantage of not fulfilling all the functions expected of a tracheal stoma.

RESUMEN

1. La cirugia desempeña un papel importante pero limitado en el tratamiento del enfermo de enfisema. La aplicación de los recursos médicos es con mucho el factor dominante para determinar el resultado final.

2. La fenestración traqueal rinde resultados que en nuestras manos no son mejores que la traqueotomía estandar. Cuando se examina la fenestración traqueal, críticamente, se le encuentra la desventaja de que no cumple con todas las funciones que son de esperarse de una estoma traqueal.

RESUMÉ

1. La chirurgie peut jouer un rôle important, mais limité chez le malade emphysémateux. De loin, c'est l'importance des mesures médicales qui est le facteur dominant pour fixer le pronostic.

2. La fenestration trachéale donne des résultats qui, entre les mains des auteurs, ne sont pas meilleurs que la trachéostomie standard. A l'examen critique, la fenestration trachéale a le désavantage de ne pas emplir toutes les fonctions que l'on attend d'un orifice trachéal.

ZUSAMMENFASSUNG

1. Operatives Vorgehen spielt eine wichtige, jedoch beschränkte Rolle bei der Behandlung von Kranken mit Emphysem. Der bei weitem dominierende Faktor, der schliesslich den Ablauf bestimmt, ist die Intensität, mit der interne Maßnahmen zur Anwendung gebracht werden.

2. Die Luftröhrenfensterung ergibt Resultate, die bei unserem Vorgehen nicht besser sind, als die standardmässige Tracheostomie. Bei kritischer Betrachtung hat die Luftröhren-Fensterung den Nachteil, nicht sämtliche Funktionen zu erfüllen, die von einer Eröffnung der Trachea erwartet werden.

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EXPERIMENTAL ABDOMINAL AORTOPLASTY

For the study, dogs have been used. In aortoplasty, jugular auto-and homografts preserved by the lyophilization method and synthetic capron and Lavsan prostheses have been utilized.

In all cases with the favorable outcome, when no circulatory disturbances in the abdominal aorta were noted clinically and roentgenologically, there appeared to be complete patency of the aorta and graft as determined by the vasography.

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Needle Biopsy of the Parietal Pleura in Tuberculous Effusion*

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Introduction

It has long been axiomatic to consider idiopathic pleural effusion in tuberculin-positive subjects to be tuberculous until proved otherwise. Until recently, proof of the tuberculous etiology has depended upon the demonstration of tubercle bacilli in the plaural fluid, but this is possible only in a minority of the cases, and confirmation of the diagnosis is usually delayed by the slow growth of the bacilli in culture.

In the last few years, diagnosis has been made through biopsy of the parietal pleura by open thoracotomy. More recently, a simpler approach with the Vim-Silverman needle has been reported[1-7] with equally good results. The needle technique is to be preferred, at least for the initial procedure, because it is simple and innocuous, convenient, repeatable, and lacks the disadvantages of a surgical procedure. The indication for thoracentesis is the indication for needle biopsy of the parietal pleura.

During the past three years, needle biopsy of the parietal pleura in cases of effusion has become common practice in the hands of interns and residents at this institution. Reports of the results in patients treated as tuberculous in smaller series of cases has been made.[4,5] The present paper includes the results in a total of 69 cases, 31 previously reported and 38 additional cases.

Method and Material

The method of biopsy is that which has been described by DeFrancis and associates.[1] Thoracentesis is done in the usual manner after procaine infiltration. A 19-gauge needle, attached to a syringe, is inserted while aspirating. When fluid is obtained, the progress of the needle is stopped and a hemostat is clamped on the needle at the skin surface. The needle is then withdrawn and the distance between its point and the hemostat is a measure of the depth of the parietal pleura. This distance, minus 5 to 10 mm., is measured from the point of the biopsy needle and marked with another hemostat. The skin is nicked with a scalpel blade and the biopsy needle is inserted to the level of the hemostat. Thus, the point of the biopsy needle will lie just outside the parietal pleura. The split needle of the Vim-Silverman assembly may then be advanced to its full depth. The outer needle is advanced about a centimeter while the split needle is held, the entire assembly is rotated 360 degrees, and removed. A small cylinder of tissue is usually found within the split needle.

Several pieces of tissue may be obtained by repeating the procedure through the same skin incision and directing the biopsy needle in slightly different directions. In the present series of patients, when the biopsy was unsatisfactory, it was repeated.

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The patients included in this series are limited to those who were treated as tuberculous and were a consecutive group of persons admitted to the Department of Pulmonary Diseases with pleural effusions and positive tuberculin tests in whom no etiology for the effusion other than tuberculosis was established. They include 24 patients with pulmonary tuberculosis and pleural effusion and 45 with pleural effusion alone.

Results

A needle biopsy was considered positive if granulomatous tissue, including caseation and epithelioid cells (usually with Langhans' giant cells), was present. Negative biopsies were required to show evidence of pleural tissue e.g., inflammatory tissue and/or fibrosis internal to skeletal muscle and/or mesothelial tissue. If this could not be seen, the biopsy was unsatisfactory. When the biopsy revealed only normal tissue, it was often repeated once or twice.

Table 1 summarizes the results of needle biopsy and pleural fluid cultures for tubercle bacilli in 45 patients who had pure effusion with no evidence of pulmonary disease compared to 24 who had, in addition to the effusion, parenchymal tuberculosis. Several cases with associated pericardial effusion are included in the first group. Several cases of miliary pulmonary tuberculosis are included in the second group.

Approximately two thirds of the patients had positive biopsies regardless of whether pulmonary tuberculosis was present or not. In contrast, there was a significant difference in the occurrence of positive fluid cultures in the two groups: 48 per cent of the patients with pulmonary disease had tubercle bacilli demonstrable in the pleural fluid whereas only 25 per cent of those with pure effusion had positive fluid cultures.

In this laboratory, the usual method of pleural fluid culture consists of direct inoculation of two tubes of Lowenstein medium and concentration of 100 ml. of fluid with sodium hydroxide followed by innoculation of a third tube of medium with the sediment. Incubation time is eight weeks, with readings at two-week intervals.

Occasional patients who had negative biopsies had positive cultures so that a small but definite increment in the etiologic diagnosis is obtained by doing both biopsy and effusion culture procedures.

If one includes all possible diagnostic methods for the establishment of a diagnosis of tuberculosis (such as sputum studies, scalene node and liver biopsies, urine cultures for tubercle bacilli, etc.) the etiology was established in 76 per cent of the pure effusion cases and in all with

TABLE 1 — STUDIES OF 69 PATIENTS WITH TUBERCULOUS OR IDIOPATHIC PLEURAL EFFUSION

	Effusion Only		Pulmonary Tuberculosis	
	No.	Per cent	No.	Per cent
Number of cases	45	100	24	100
Tubercles on needle biopsy of pleura	30	67	17	71
Tubercle bacilli in culture of effusion	9*	23	11**	48
Positive biopsy and/or effusion culture	32	71	20	83
Tuberculosis proved by diagnostic methods	34	76	24	100
Idiopathic pleural effusion	11	24	0	0

*of 39 cases
**of 23 cases

both effusion and pulmonary disease. The high rate for the latter group was due to the fact that sputum was usually positive for tubercle bacilli.

However, there remain 11 cases in the pure effusion group in which an etiologic diagnosis was not established by any means and these constitute instances of "idiopathic" pleural effusion. Because their tuberculin skin tests were positive, they were considered tuberculous and treated as such.

The wisdom of such an attitude is bolstered by the fact that a single negative biopsy by no means rules out tuberculosis. Of 27 patients in the entire series of 69 who had negative single biopsy, 12 had a second biopsy and three were positive for tubercles. Three patients who had two negative biopsies were biopsied a third time and two of these were positive. One had three negative biopsies, but had positive fluid culture for tubercle bacilli. One who had a single negative needle biopsy was subjected to open biopsy of the pleura and this tissue showed tubercles. Therefore, negative needle biopsies do not completely exclude tuberculosis as the etiology of pleural effusion.

Although the amount of tissue obtained with the Vim-Silverman needle is small, Ziehl-Nielsen stain of the tissue may sometimes reveal acid-fast rods. The tissue may also be cultured for tubercle bacilli; this was done in two cases and culture was positive in one.

In three cases with persistent fluid, the needle biopsy was repeated some time after the positive initial biopsy during therapy. In one patient, tubercles persisted after one month of antimicrobial treatment while in two cases, tubercles were no longer demonstrable after three months of specific therapy.

The only untoward reactions of needle biopsy in this series of patients were two instances of traumatic pneumothorax. These occurred in a total of 88 biopsies. Therefore, the risk of the procedure is quite small. Both cases of pneumothorax were small and required no special management. There were no instances of bleeding or infection of the needle track. No particular pre- or post-biopsy precautions were taken.

Discussion

The results of this series indicate that needle biopsy of the parietal pleura is a valuable procedure in the etiologic diagnosis of tuberculous pleural effusion. Any method that will augment the diagnostic yield in "idiopathic" pleural effusion is of increasing importance in view of the fact that tuberculosis is becoming more common in older men. It is in this segment of the population that the differential diagnosis between tuberculous effusion and carcinomatous effusion is a prime consideration. Needle biopsy often provides an early definitive diagnosis.

Needle biopsy of the parietal pleura has also been of frequent value in the diagnosis of malignant effusion, but in such cases cell block of the pleural fluid has usually revealed the diagnosis. Although one procedure may be positive when the other is negative in cases of carcinoma, needle biopsy finds its greatest usefulness in the diagnosis of tuberculous effusions because it is much more often positive than is culture of pleural fluid and because the result is obtained much more quickly.

The specificity of caseating tubercles may be questioned, since these lesions are on rare occasions produced by agents other than tubercle bacilli. However, nontuberculous causes are sufficiently uncommon so that the error in diagnosis is quite small in patients who are tuberculin reactors.

It must be emphasized that, if the needle biopsy is unrevealing on one occasion, it must not be assumed that either tuberculosis or carcinoma has been excluded. This is particularly true in cases of carcinoma, since the implantation of malignant tumor on the parietal pleura is sometimes not as diffuse a process as seems to occur in tuberculosis of the pleura. A second or third needle biopsy may be more rewarding. If not, then open pleural biopsy may be considered. In the meantime, all diagnostic

methods should be brought to bear on the case of effusion because pleural fluid cultures, sputum cultures, or biopsies of other tissues may yield a specific diagnosis when the pleural biopsy is negative.

The frequency with which actual tubercles can be demonstrated in the parietal pleura lends support to the idea that tuberculous pleural effusion is largely due to infection of the pleura rather than to allergic reaction.

SUMMARY

Needle biopsy of the parietal pleura was done from one to three times in 69 patients with pleural effusion treated as tuberculous. This series included 45 cases of pure effusion without pulmonary disease and 24 cases of effusion with pulmonary tuberculosis. Biopsy proved the tuberculous etiology in two thirds of the patients, whereas pleural fluid cultures were positive for tubercle bacilli in 23-48 per cent of the patients. Tuberculosis was established as the etiology in all but eleven cases by all methods of histologic and bacteriologic approach.

Needle biopsy was the single most rewarding diagnostic procedure used in this series of patients. It is simple, convenient, safe and repeatable.

RESUMEN

Se realizó la biopsia por medio de aguja de la pleura parietal por una a tres veces en 69 enfermos con derrame pleural que se trataba como tuberculosis.

Esta serie incluye 45 casos de derrame puro sin enfermedad pulmonar y 24 con tuberculosis pulmonar.

La biopsia demostró la naturaleza tuberculosa en dos tercios de los enfermos en tanto que el cultivo de líquido pleural fué positivo en 23-48 por ciento de los enfermos. No se estableció la etiología tuberculosa en 11 casos siendo comprobada en los restantes por estudio histológico o bacteriológico.

La biopsia por aguja resultó el procedimiento simple que dió mejores resultados diagnósticos en esta serie. Es sencilla, conveniente, segura y puede repetrise.

RESUMÉ

Une biopsie de la plèvre pariétale à l'aiguille fut faite en un à trois temps chez 69 malades, atteints d'épanchement pleural traité comme tuberculeux. Ce groupe comprensit 45 cas d'épanchement pursans affection pulmonaire et 24 cas d'épanchement avec tuberculose pulmonaire.

La biopsie démontra l'étiologie tuberculeuse chez deux tiers des malades, alors que les cultures du liquide pleural ne furent positives pour le bacille tuberculeux que chez 23-48% d'entre eux. L'étiologie tuberculeuse fut établie chez tous les malades sauf II cas par toutes les investigations histologiques et bactériologiques.

La biopsie à l'aiguille est le seul procédé de diagnostic utilisé dans ce groupe de malades. Elle est simple, souhaitable, sans danger et peut être répétée.

ZUSAMMENFASSUNG

Eine Nadel-Biopsie der parietalen Pleura wurde ein-bis dreimal bei 69 Kranken mit als Tuberkulose behandeltem pleuralen Erguβ vorgenommen. Diese Reihe von Patienten enthalt 45 Fälle von reinem Erguβ ohne pulmonale Erkrankung und 24 Fälle von Erguβ mit Lungentuberkulose.

Die Biopsie bewise die tuberkulöse Ätiologie in zwei Drittel der Kranken, wohingegen die Kulturen der pleuralen Erguβe auf Tuberkelbazillen in 23-48% Fällen positiv waren. Eine Tuberkulose als Ursache wurde mit 11 Ausnahmen in allen Fällen gesichert durch beide Methoden, nämlich auf histologischem und bakteriologischem Wege.

Die Nadel-Biopsie ist als einzelne Methode das am meisten lohnende Verfahren, das bei dieser Reihe von Patienten zur Anwendung kam. Sie ist ainfach, gut verträglich sicher und läβt sich wiederholen.

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Nitrites in the Treatment of Bronchial Asthma*

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Spasm of the smooth muscle of the bronchi and bronchiole, edema of the mucosa leading to mucous collection, and occlusion of aveoli probably cause most of the symptoms of asthma. This resultant bronchial inflammation and obstructive emphysema may be acute, recurrent, or chronic. The degree to which these changes interfere with the passage of respiratory gases determined the severity of the condition. Frequently, one is unable to uncover the causative factors of these anatomic and physiologic changes. Many times, even when the causes are known, the difficulty of eradicating these factors has made symptomatic therapy the standby for the treatment of bronchial asthma.

For many years, the sympathomimetic drugs have been the foundation upon which the majority of bronchial asthmatic remedies have been built. Epinephrine was modified to form isopropylarterenol in the search for drugs with greater ease of administration, prolongation of

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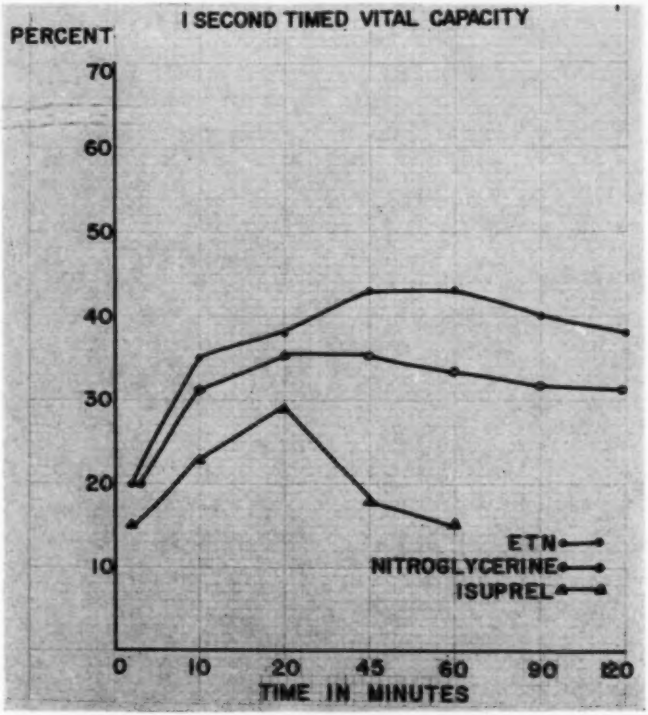


TABLE 1 — 1 SECOND TIMED VITAL CAPACITY
O.6 Mg. NITROGLYCERINE-SUBLINGUAL
(Per cent)

No.	Name	Age - Sex - Race	Pre-Med	10 Min.	20 Min.	45 Min.	60 Min.	90 Min.	Remarks
1.	C. D.	26 M W	48	70	75	81	75	60	No side effects
2.	R. L.	28 F W	54	50	48	67	57	—	No side effects
3.	B. M.	52 F W	45	50	55	45	52	43	No side effects
4.	B. A.	33 F C	61	50	52	—	—	—	Discontinued
5.	R. S.	43 F C	56	57	60	63	52	—	No side effects
6.	P. A.	47 F W	66	75	74	77	87	77	No side effects
7.	S. G.	33 M W	56	60	54	—	80	—	No side effects
8.	Brown	42 F C	61	68	52	54	60	—	No side effects
9.	P. G.	46 F C	32	40	40	43	45	—	No side effects
10.	J. S.	34 M C	42	61	66	—	72	43	No side effects
11.	Y. B.	27 F C	50	52	40	60	—	—	Cooperation poor

effect and milder side reactions. Ephedrine was also employed for this purpose. The xanthines came into use because of their ability to relieve smooth muscle spasm. All of these drugs are effective in various degrees, but resistance develops to each and side effects may limit the quantities used. One attempt to overcome the resistance that the patient develops to the sympathomimetic drugs was the utilization of the drugs in combination with ganglionic blocking agents.[1] This is of some help, but the

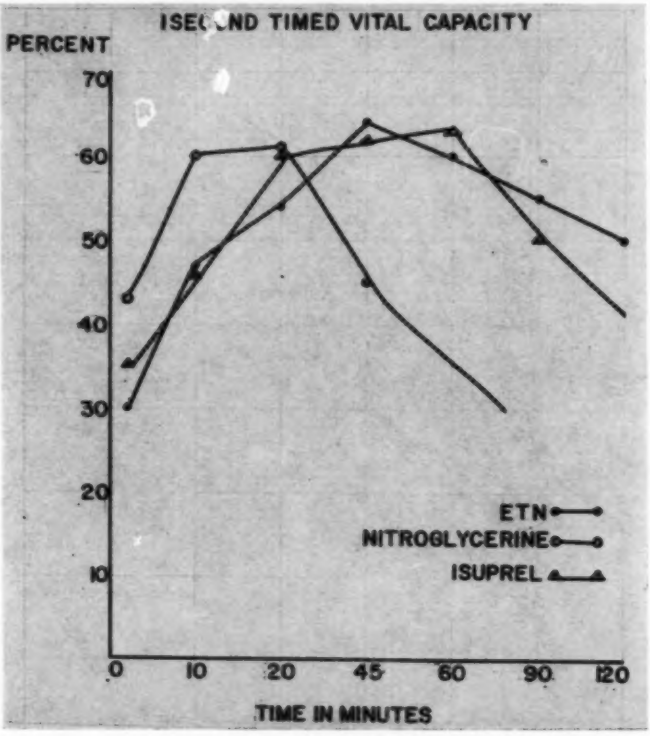
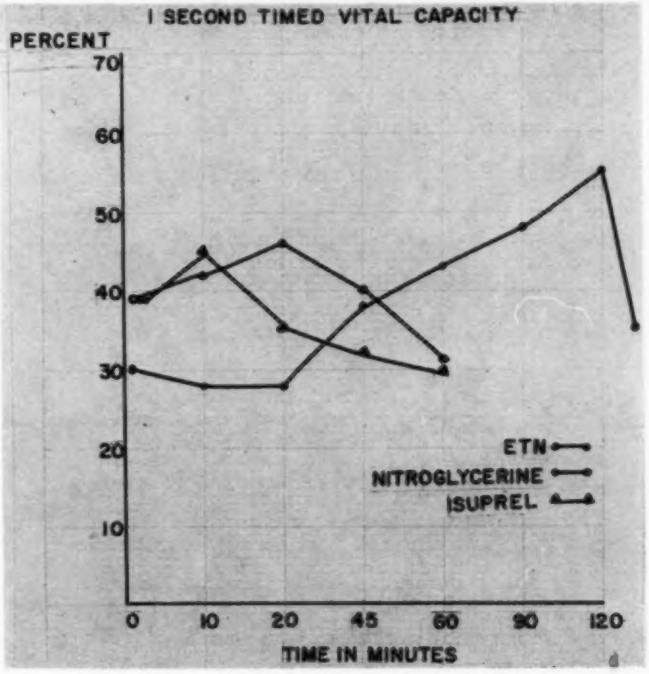


TABLE 2 — 1 SECOND TIMED VITAL CAPACITY

Case No.	Pre-Med.	10 Min.	20 Min.	45 Min.	60 Min. Sublingual	6 Days Oral
1.	40 per cent	42 per cent	50 per cent	66 per cent	66 per cent	83 per cent
2.	44 per cent	60 per cent	60 per cent	62 per cent	60 per cent	75 per cent
3.	40 per cent	45 per cent	60 per cent	65 per cent	65 per cent	0 per cent
Erythrol Tetranitrate — 15 mg.						(T.I.D.)

potency of the ganglionic blocking agent used is limited by the undesirable side effects or rather extension of action of the drugs. Steroids have been used during the past decade, but here again, dangerous side effects appear with increased dosage and/or prolonged administration. An additional problem is the reexacerbation of the disorder if withdrawal of the drug is too abrupt. Expense of the medication may at times be a problem. Meperidine hydrochloride is resorted to by Segal[2] when other measures fail or fastness to epinephrine or to the xanthines has developed. This cannot be recommended as a routine measure, however, and too large or too frequent a dose may depress respiration.

Ideally, bronchodilation should be the only effect of a drug used for the symptomatic relief of bronchial asthma. Bronchodilatation leads to an increased rate and depth of respiration. Oxygen intake is augmented, and the level of oxygen consumption of the tissue cells elevated, producing greater tissue oxygenation.[3] The relaxing action of the nitrites on smooth muscle was recognized and exploited to effect bronchiole dilatation early in the modern era of medicine.[4] In addition to sublingual



administration, early methods included the inhalation of the fumes produced by the burning of potassium nitrite combined with stramonium, and by burning paper impregnated with saltpeter.[5] Both of the remedies caused the reduction of nitrate to nitrite in the fumes to be inhaled. Ease of administration was certainly not one of the virtues of such means of medication. About the end of the first decade of this century these drugs gradually lost favor and fell into disuse.

Johnson *et al.*,[6] noting the effectiveness of sublingual nitroglycerine in the relief of the respiratory distress of paroxysmal nocturnal dyspnea, attempted to determine the response of the drug on pulmonary artery pressures in patients with failure of the left ventricle. In a physiologic study using right heart catheterization, nitroglycerine was shown to produce a prompt reduction in the pulmonary artery hypertension associated with failure of the left ventricle.

When we applied these results clinically to patients in varying degrees of congestive failures, it was observed that the cases of cor pulmonale associated with chronic bronchial asthma had the greatest relief of respiratory distress when treated with nitroglycerine. It was postulated that the nitrite had a two-fold action both in reducing the pulmonary artery hypertension and relieving the concomitant bronchial spasm. Utilizing plethysmographic methods[7,8] for evaluating nitrites used in the treatment of cardiovascular disease, we found that nitroglycerine

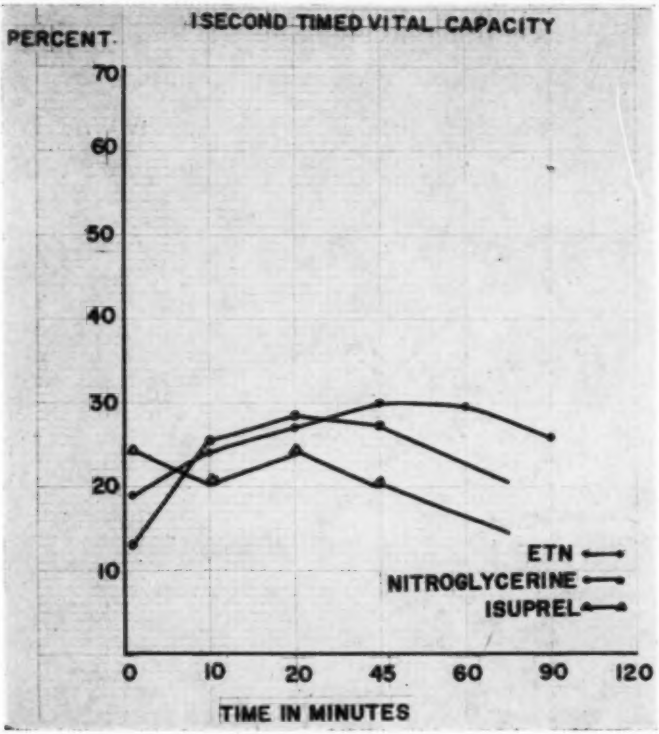
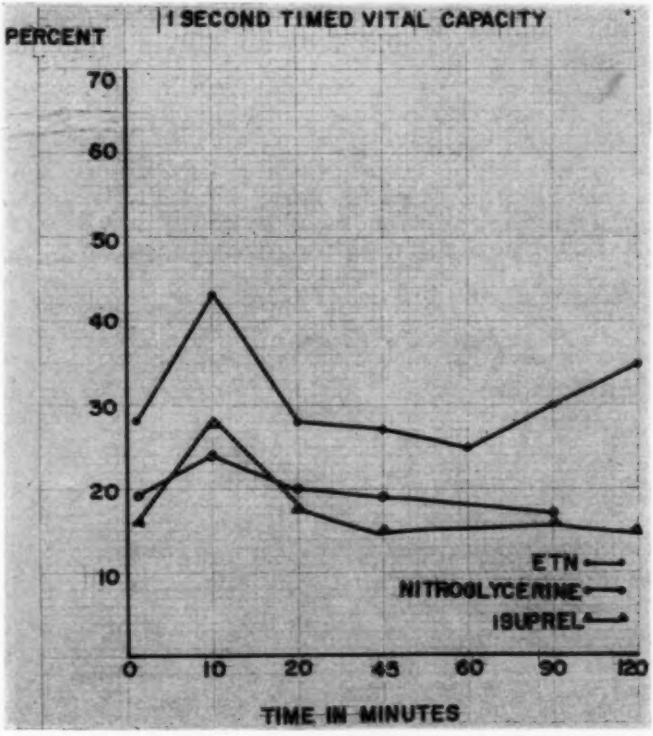


TABLE 3 — EFFECT OF ROUTE OF ADMINISTRATION—NITROGLYCERINE 0.6 mg.
1 SECOND VITAL CAPACITY — Per cent

Case No.	Pre-Med.	10 Min.	20 Min.	45 Min.	60 Min.
1 Sublingual	43	58	58	—	33
1 Oral	38	52	62	63	70
2 Sublingual	20	33	41	44	48
2 Oral	28	30	50	46	50

and erythrol tetranitrate were as effective orally as sublingually in potency and duration of their vasodilating effect. Onset of action was little affected by the different modes of administration.

With these observations in mind, it was thought feasible to conduct a study of the effects of nitroglycerine as a short acting nitrite, and erythrol tetranitrate as a long acting nitrite, upon patients with bronchial asthma. Isopropylarterenol was also included in the evaluation, since it is a widely used and accepted epinephrine derivative, and in that capacity might serve as a yardstick against which to compare the therapeutic effectiveness of the nitrites.

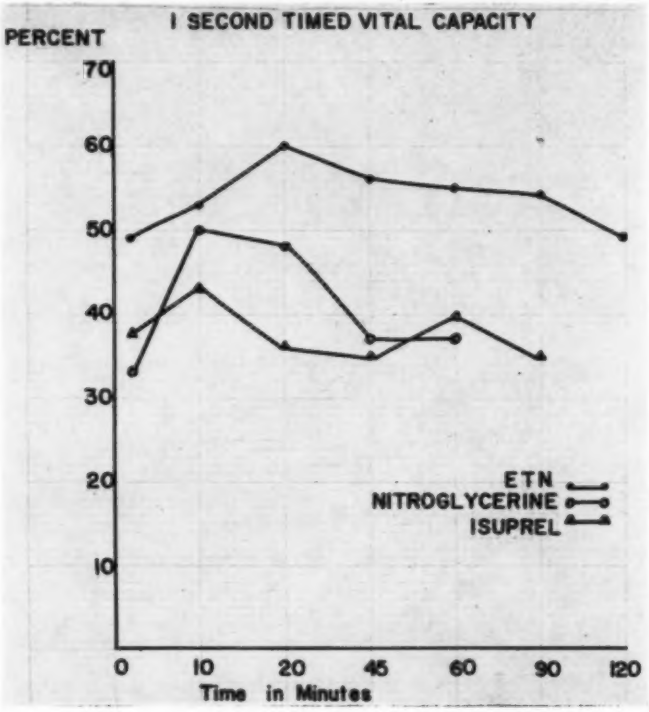


Method

The purposes of testing pulmonary function in bronchial asthma are to determine the degree of disability of the patient, to follow the progress of the disease in individual cases, to provide a means of direct testing of the pulmonary tree with suspected allergens, and to assay the effectiveness of various therapeutic drugs.[9,10]

The tests of pulmonary function employed in bronchial asthma include the vital capacity, timed vital capacity and index of intrapulmonary mixing of gases as determined by the open circuit or closed circuit method.

The vital capacity is the maximal volume of gas that can be expired from the lungs by forceful effort after a maximal inspiration. It may be normal or decreased in patients with bronchial asthma as compared with the predicted normal.[11] Cournand[12] described a method of graphic registration of breathing by means of a modified basal metabolism apparatus and noted that change in the form of deep breathing, particularly retarded expiration, revealed early stages of pulmonary emphysema or bronchial asthma. Gaensler[13] found that timed vital capacity measurements provide a method for indicating marked slowing of the expiratory phase commonly found in both these conditions. In the presence of airway obstruction or impaired elastic recoil, small reductions of total vital capacity are accompanied by marked decrease of timed capacities. Unlike the vital capacity, the time capacities correlate well with maximal breathing capacity, air velocity index, and the ratio of residual volume to total lung capacity.



The maximal breathing capacity is not a practical method when one is desirous of evaluating the onset and duration of action of a drug since it is a tiring procedure and therefore cannot be repeated at closely spaced intervals. It is an excellent method when employed to gauge the long term response of a patient to a drug and was thusly employed in our study. To determine the onset, maximum response and duration of action of the drugs used in this study, we employed closed circuit spirometry for graphic recordings when it was feasible to bring the patient to the apparatus. When it was easier to bring the apparatus to the patient, we used a simple attachment to the ordinary dial type vital capacity machine[4] which automatically records the total vital capacity and the fraction of the total volume exhaled during the first second of the maximal expiratory effort. Normal persons are capable of exhaling 83, 94 and 97 per cent of the total vital capacity during one, two and three seconds.

Results

Nitroglycerine 0.6 mg. was administered sublingually to 11 patients during acute episodes of bronchial asthma. Three of these were unresponsive to intravenous aminophylline 0.5 gm. given one and a half hours prior to nitrite therapy. Their ages ranged from 26 to 52 years, eight women and three men. One second timed vital capacities were recorded by means of a simple timing attachment to the ordinary dial type vital capacity machine prior to medication and 10, 20, 45, 60 and 90 minutes following. In all cases except one, who refused to perform the function tests, there was improvement in the one second vital capacity. All 11 patients showed subjective improvement. (Table 1)

Three patients with bronchial asthma varying from six to 18 years were given erythrol tetranitrate* 15 mg. sublingually and tested by the same method. A one second timed vital capacity was performed prior to medication and 10, 20, 45, and 60 minutes following. They were then placed on a regimen of erythrol tetranitrate 15 mg. orally three times a day for six days. Increased timed vital capacity and subjective improvement was noted in all. (Table 2)

Two patients were given nitroglycerine 0.6 mg. sublingually and the one second vital capacities were recorded by closed method spirometry. This was later repeated with orally administered nitroglycerine. These results (Table 3) indicate that the two methods of administration have equal therapeutic value.

A 51 year-old white man with old pulmonary tuberculosis was referred to us for pulmonary function tests. It was found that his predicted maximal breathing capacity was 47 per cent of normal. He was placed on a regimen of breathing exercises for

TABLE 4

Case Report — Date	8-20-58	10-2-58	10-27-58
Vital Capacity — Total	2472 cc.	2400 cc.	- - - - -
Per cent Predicted	67	65	- - - - -
1 Second V.C. — Per cent	45	30	- - - - -
M.B.C. Liters/Min.	44	50	64
Per cent Predicted	47	50	67.6

*Supplied as Cardilate,® Burroughs Wellcome and Company, Inc., Tuckahoe, New York.

TABLE 5

Drug	Onset Time	Maximum Effect	Duration of Action	Side Effects
Erythrol Tetranitrate 15 Mg. Sublingual	10 Min.	45 Min.	1-3 Hours	None
Nitroglycerine 30 Mg. Sublingual	5-10 Min.	20 Min.	45-90 Min.	None
Isuprel 15 Mg. Sublingual	5-10 Min.	20 Min.	20-45 Min.	None

a one month period. At the end of this time his predicted breathing capacity had increased to 52 per cent of normal. He was then given erythrol tetranitrate 15 mg. three times a day orally for six days. Following this course of therapy his predicted maximal breathing capacity increased to 67.6 per cent of normal. (Table 4) Lobectomy was then successfully performed.

Six patients were evaluated with erythrol tetranitrate 15 mg., nitroglycerine 0.3 mg., and Isuprel 15 mg. by closed spirometry methods and the one second timed vital capacities determined from the graphic recordings. Figures 1 to 6 illustrate the results in each patient. Time of onset of action usually began within 10 minutes with all three drugs. The maximum effect averaged 20 minutes for nitroglycerine and Isuprel and 45 minutes for erythrol tetranitrate. Duration of action was longest for erythrol tetranitrate. (Table 5)

Discussion

The ideal drug for the symptomatic relief of bronchial asthma should have the attributes of ease of administration, potency of action, duration of effect, absence of untoward reaction, economy and the failure to build resistance to the medication. Our results with nitroglycerine and erythrol tetranitrate indicate that oral administration is feasible. The potency of the nitrites used in this study compare favorably with isopropylarterenol (Isuprel), an accepted epinephrine derivative used in the treatment of bronchial asthma. Duration of effect is definitely superior for erythrol tetranitrate. Undesirable side effect of the nitrites are minimal and have been known and studied for years. Resistance to nitrites as used for angina pectoris has never been much of a therapeutic bulwark. We believe that this investigation indicates a place for the nitrites in the armamentarium of drugs used to treat bronchial asthma.

We have used these nitrites on a limited scale for the treatment of congestive heart disease because of their smooth muscle relaxing effect and their ability to lower pulmonary arterial pressure. Our results to date are gratifying enough to indicate the desirability of an expanded investigation into this field.

SUMMARY

1. Nitroglycerine and erythrol tetranitrate are effective drugs for the symptomatic treatment of bronchial asthma.

2. The potency and duration of action of these nitrites surpass that of isopropylarterenol.

3. Sublingual administration has no advantage over oral administration.

RESUMEN

1. La nitroglicerina y el tetranitrato de eritrol son efectivas para el tratamiento sintomático del asma bronquial.

2. La potencia y la duración de acción de estos nitritos sobrepasa la del isopropilarterenol.

3. La administración sublingual no tiene ventajas sobre la administración oral.

RESUMÉ

1. La nitroglycérine et le tetranitrate d'érythrol sont des produits efficaces dans le traitement symptomatique de l'asthme bronchique.

2. La puissance et la durée d'action de ces nitrates surpasse celle de l'iso-propylarterenol.

3. L'administration sublinguale n'a auçun avantage sur l'administration buccale.

ZUSAMMENFASSUNG

1. Nitroglyzerin und Erythol-Tetranitrat sind wirksame Arzneimittel für die symptomatische Behandlung des Bronchial-asthma

2. Stärke und Wirkungsdauer dieser Nitrite übertreffen diejenige von Isopropylarterenol.

3. Gegenüber der oralen Anwendung bietet die sublinguale keine Vorteile.

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ANGIOPNEUMOGRAPHY IN SOME PULMONARY AND MEDIASTINAL DISEASES

Angiopneumography was used in 60 patients affected by lung cancer, various chronic inflammatory lung processes and some mediastinal diseases. In addition, vessel injection of 20 lung preparations removed because of the aforementioned diseases has been made. In lung cancer, angiopneumography reveals poor or no vascularization in the pathologic area. The technic of selective angiopneumography is the best to reveal vascular changes. Angiopneumography was found to be of value in determining signs of inoperable lung cancer.

In chronic lung suppuration, angiopneumography is of no diagnostic value and aids but little in the differential diagnosis of malignant pulmonary growths.

In unresolved pneumonia, there is normal or increased vascularization. Angiography helps to make diagnosis of the aneurysm of mediastinal vessels.

Gachja, P.: "Angiopneumography in Some Pulmonary and Mediastinal Diseases," *Chest Surgery (USSR)*, 85:39, 1960.

PROPHYLACTIC ACTION OF INH AGAINST WHOOPING COUGH

The authors have observed that INH exerted evident prophylactic influence in respect to whooping cough during an epidemic in a group of children who were given this drug for the prevention of tuberculosis.

Rustichelli, V., Mastantuono, C., and Palumbo, F.: "Prophylactic Action of INH Against Whooping Cough," *Gior. Med. e Tisiol.*, IX:28, 1960.

Bronchopulmonary Sarcoidosis Confirmed by Bronchoscopic Biopsy

A Report of Two Cases with a Review of the Gross and Histologic Descriptions of All 28 Previously Recorded Cases in the Literature

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It is less than a score of years (1941)[1] since the first confirmatory bronchial biopsy was obtained from a case of pulmonary sarcoidosis. The literature concerning means of effecting a diagnosis in this pleomorphic, often systemic disease, is voluminous. In three large series including a combined study from the Johns Hopkins Hospital and the Massachusetts General Hospital,[3] a series by Riley[4] from New York City and one from the Veterans Hospital,[10] totaling 321 cases, only one positive bronchial biopsy compatible with a sarcoid lesion was described. This case was the first described in the literature.[1] It is curious that with hilar lymph node involvement and parenchymal changes listed in the majority of the cases, only one other case even mentions bronchoscopic abnormalities[4] in these three series, without substantiation of biopsy. In the series reported by Riley,[4] 6 of 52 cases (14 per cent) had symptoms of wheezing, suggestive of stenotic bronchial processes. This alone would indicate that with routine bronchoscopy and biopsy the yield of positive biopsy material would have been substantial in the 45 of Riley's 52 cases presenting pulmonary involvement. It is known that sarcoid changes in the respiratory mucosa occur more frequently than is generally realized. A positive laryngeal biopsy was obtained relatively early.[2,5] It was not until Kalbian[14] made a routine bronchoscopic study on 11 known cases of sarcoidosis, finding abnormalities in nine and positive biopsies in three cases, that the significance of bronchial biopsy as a source of diagnostic material was realized. In the Dunner *et al.*[17] series of 109 cases, 131 positive biopsies were obtained from various sites, namely, 56 per cent from lymph nodes, 15 per cent from the liver, 12 per cent from the lung by needle biopsy, 9 per cent from the skin and 8 per cent from other sites. Bronchial biopsies were not mentioned in spite of a high percentage of lung and hilar lymph node involvement, more than any other organic system.

The following two cases (1956-57 and 1959), with histories and illustrations, are presented with suggestive findings on bronchoscopy and the positive diagnostic evidence obtained through biopsy material.

Case 1: I.M.C., a white woman, age 28, was first seen in August, 1956. Her illness began in mid-1954 when she developed wheezing respiration accompanied by dry cough. Early in 1956 she was referred to a chest clinic where a chest x-ray film was taken and she was told that she had tuberculosis. Sputum studies were negative by culture and guinea pig inoculation. She was placed immediately on streptomycin twice weekly and PAS daily. An x-ray film of her chest on August 7, 1956 showed a fairly dense, somewhat rectangular parenchymal infiltration in the right upper lung field, bilaterally enlarged hilar nodes, and a fibrous infiltrate in the left subapical region with a general increase of the markings in the lower lung fields. Bronchoscopic examination was made at the Maryland General Hospital on September 11, 1956. The findings were a foreshortened, broadened and edematous carina without the usual

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sharp spur. There was mild edema of the right bronchus and the telescopic view of the upper lobe of this bronchus showed a thickened projecting margin, with several round projections seen particularly on the inferior and anterior circumferences. The lumen was narrowed to one-half its normal size and only the posterior division of the bronchus could be seen. From it exuded some bubbly purulent material. An attempt at biopsy was made and several small shreds of tissue were obtained from the rather firm margin of the upper lobe of the right bronchus. The left bronchus was examined and except for slight edema of the left basilar divisions and a somewhat broadened left upper lobe divisional spur, no gross abnormality was detected. At the time it was felt that there was stenosis of the upper lobe branch of the right bronchus and mildly allergic (asthmatoid) bronchial mucosa, as suggested by the edema and spasm of both bronchi. The biopsy material proved insufficient for diagnosis and was described by the pathologist as showing tiny shreds of epithelium, with a large amount of fibrin and a blood clot. Bronchoscopic secretions were reported negative for tubercle bacilli by culture and guinea pig inoculation, also by the three gastric washings which were done during this hospitalization. She was then placed on isoniazid and PAS instead of streptomycin. She was followed until early 1957. After another chest x-ray film (Fig. 1) she was admitted to Church Home and Hospital on January 29, 1957. This chest film showed an increase in the amount of infiltration in the right and left upper lobes with persistence of the hilar adenopathy. Upon admission, the pertinent history was the same as previously reported, with emphasis on the absence of tuberculosis in the family. Physical examination revealed a fairly well developed, well nourished, slightly obese woman. Her vital signs were normal except for a temperature of 99.2°F. The physical examination proved negative except for positive findings in the chest. The lungs showed dullness in the right upper part of the chest and increased tactile fremitus at the left interscapular area and the left base. Bronchial breath sounds were heard over the left upper lung field and inspiratory wheezes bilaterally. Rales were not elicited on either side. Blood cell counts and chemistries including serum calcium and A/G ratio, as well as blood sugar and NPN were essentially normal. The sputum was negative for acid-fast bacilli. A second strength PPD tuberculin test was reported negative after 48 hours. This was repeated and again was negative. Histoplasmin skin tests were negative on two occasions. On January 30, 1957, a second bronchoscopy was performed and revealed the following: Secretions were seen coming from the lower

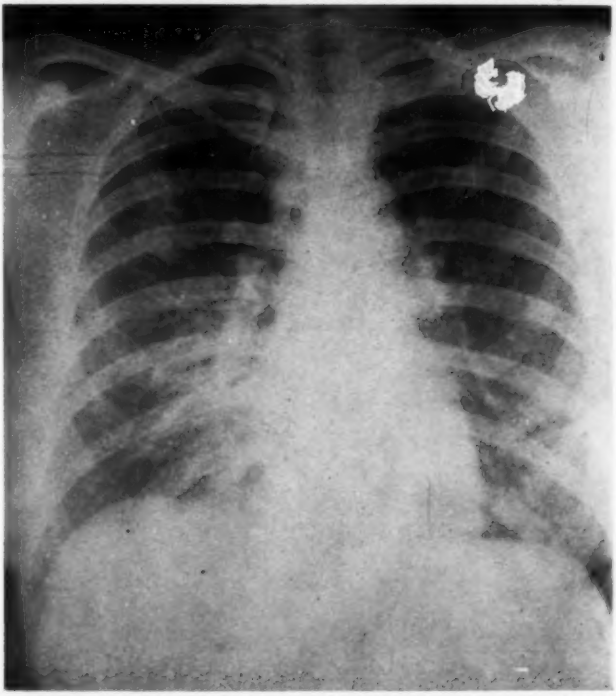

FIGURE 1: Chest roentgenogram January 19, 1957, after 4 months of streptomycin-isoniazid therapy and prior to admission to Church Home and Hospital. Evidence of increase in the upper lobe infiltrations as well as the size of the hilar lymph nodes as compared with pre-admission findings.

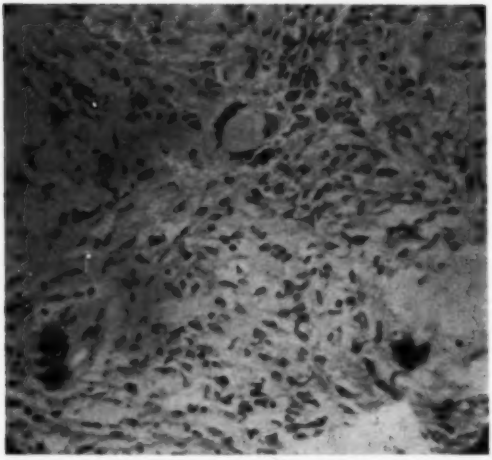

FIGURE 2: Histology of bronchoscopic biopsy specimen, showing non-caseating epithelioid cell follicles with giant cells.

lobe of the left bronchus, which was moderately irritated and considerably contracted by edema. The mucosa of this lower lobe had a velvety appearance and the lumen of the intermediary bronchus was reduced by 50 per cent. This reduction was even more noticeable below the superior segment branch and extended into the basilar divisions. The velvety red mucosa was friable and hemorrhagic secretions were obtained as a result of the instrumentation. Right angle telescopic visualization at this time showed the right upper lobe orifice fixed with a triangular contracted outline. On the inferior and anterior portions of its margins there was a whitish scar, probably the result of the previous biopsy attempt. The right upper lobe bronchus was definitely elongated and still reduced by 50 per cent with only 1 subdivisional spur recognizable. A biopsy was taken from the velvety mucosa of the lower lobe of the left bronchus, namely a basilar divisional spur. The bronchial biopsy (Fig. 2) on pathologic section was interpreted as chronic granulomatous tuberculoid reaction of the bronchial mucosa, compatible with the diagnosis of sarcoidosis. During her hospital stay, she was placed on steroids (prednisone, 40 mgm. daily), as well as isoniazid (100 mgm. daily). Some objective improvement occurred. The cough decreased and wheezing disappeared by the time she was discharged on February 21, 1957. She remained on Prednisone for six weeks, followed by maintenance doses of 15 mgm. daily for 10 months. A chest film after seven weeks of steroid therapy showed slight improvement. During the 10 months of steroid maintenance she complained of loss of libido, epigastric distress, weight gain and irritability. Prednisone therapy was discontinued after 10 months, when it was felt that maximum benefit was obtained, and she became free of com-

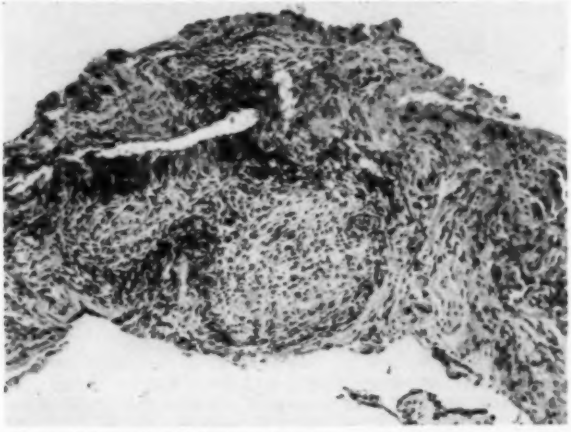

FIGURE 3: Second bronchial biopsy, showing granulation type tissue and fibrosis.

pliants. However, there was a moderate increase in the roentgenographic findings throughout 1958. When this became more noticeable in early 1959, a re-appraisal was deemed necessary and she was admitted to the Maryland General Hospital in February of 1959. A third bronchoscopy was performed on March 2, 1959. As seen telescopically, the right upper lobe orifice again showed a stenosis of 50 per cent with considerable elongation and over-hanging of a whitish, fibrotic anterior margin. No longer could any portion of the subdivisoins be seen. The firmness of the margin made it impossible to attempt a biopsy from this region. The left bronchus was then examined and showed stenosis of about 40 per cent of the intermediary left lower lobe bronchus. The individual basilar divisions, of which only two could be seen, were stenosed by more than 50 per cent. The mucosa no longer showed the previous velvety red appearance, but was whitish, pale, edematous with a tendency to fibrous contraction. Again a biopsy attempt was made from the left basilar divisions and three small shreds of mucosa were obtained. Pathologically, this second bronchial biopsy (Fig. 3) no longer showed specific sarcoid tissue although the granulomatous pattern could still be recognized. The tissue showed partial fibrosis and was considered to represent nonspecific granulomatous mucosa. On March 6, 1959, a right cervical gland biopsy was done, which histologically showed typical sarcoid follicles, non-caseating, consisting of epitheloid cells as well as giant cells. Schaumann bodies could be demonstrated within the giant cells. Sputa and gastric washings were negative by smear and culture and the histoplasmin and tuberculin skin tests, which were repeated again proved negative. It is interesting to note, however, that the bronchial secretions grew out a single colony of acid-fast bacilli, which on culture and guinea pig inoculation proved to be virulent tubercle bacilli. She has remained asymptomatic since, with little or no change in the x-ray films. It has not been possible to demonstrate tubercle bacilli again in any of her secretions. However, a prophylactic course of isoniazid is being continued. This relates to the concept of Scadding and Citron[13] who believe in a close relationship of sarcoidosis with tuberculosis.

Case 2: J.A.S., a 20 year-old white woman, was first seen on July 3, 1959, when her chest x-ray film (Fig. 4) showed suggestive changes of pulmonary sarcoidosis. She was admitted to Church Home and Hospital on July 9, 1959 with the chief complaint of intermittent chest pain of about 18 months' duration associated with progressive shortness of breath unrelated to effort. The family history was unrevealing except for the death of one sister at the age of 23 of carcinoma of the stomach. The past history was interesting since she is said to have had five attacks of pneumonia before the age of 10. Married for four years, she had one pregnancy resulting in a healthy child. She worked for a manufacturer of plastics, but insisted that she had not been in contact with any toxic airborne substances. A chest x-ray film in mid-1958 was

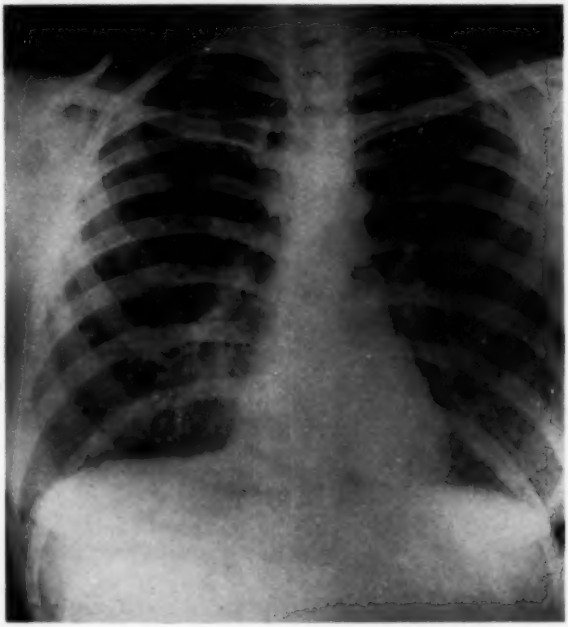

FIGURE 4: Case 2, Initial chest roentgenogram July 3, 1959, showing diffuse infiltrations of small patchy and streaky areas throughout both lung fields.

said to have shown "spots on both lungs." On the basis of a second chest film in March of 1959 at a chest clinic, the probable diagnosis of pulmonary tuberculosis was considered and she was so informed. Sputum examinations were carried out and reported negative for tubercle bacilli by culture. The vital signs were normal; the temperature was 99°F. The physical examination of this well developed, slightly obese female was essentially negative except for moderately harsh breath sounds and an occasional muffled wheeze, heard bilaterally. Laboratory data: The urine was free of sugar and albumin. Hematologic findings were within normal limits. Blood chemistries showed: Blood sugar 95, NPN 28, total proteins 8, albumin 5.3, calcium 10.2, phosphorus 3.8, sodium 145 m.Eq./1, potassium 4.9 m.Eq./1. The A/G ratio was 1.9. Alkaline phosphatase was 3.3 units, thymol turbidity 0.3 units. There were no LE cells seen. The Fishberg concentration test and the electrocardiogram were normal. A chest x-ray film following admission showed that there were diffuse changes seen throughout both lung fields, consisting of fine linear and interlacing densities, with superimposed small nodular lesions. The changes were more pronounced on the right than on the left and extended peripherally from both hili. The cardiovascular structures were found to be normal. The radiologist's impression was: Bilateral pulmonary changes which were most likely caused by sarcoidosis. Upon x-ray examination of the paranasal sinuses an unusual incidental finding in the skull was observed. Diagnostic skull x-ray films showed two spherical areas, 1.5 cm. in diameter, of mottled calcification within the cranial cavity. They were symmetrically placed, and therefore from their location they were presumed to represent calcification in the gloma of the chorioid plexuses. Their significance at this time was doubtful, but the possibility of a disturbed calcium metabolism in the past raised interesting questions in relation to similar disturbances seen in some cases of sarcoidosis. A second strength PPD tuberculin test was done as well as a histoplasmin skin test. Both were reported negative after forty-eight hours. The tuberculin test was repeated after several days, resulting in a negative response again. The hospital course was as follows: On July 13, 1959 the patient was bronchoscoped and a bronchial biopsy was taken. The bronchoscopic report showed the following highlights: The carina was broadened, thickened and blunted. The right upper lobe orifice showed moderate thickening of the mucosa; the right middle lobe orifice also was thickened and showed edema. The right lower lobe bronchus showed considerable edema with projection of the edematous mucosa anteriorly. The color of the mucosa was whitish and pale. Seen telescopically, the left bronchus showed minimal edematous thickening of the upper lobe divisional spur and some edematous changes in the basilar divisions. A bronchial biopsy was obtained from the projecting anterolateral portion of the right lower lobe bronchus, near the basilar divisions. Histologically, the bronchial biopsy (Fig. 5) showed within the submucosal zones several structures of tubercle-like origin, revealing a layer of epitheloid cells irregularly arranged. According to the pathologic report, the appearance was suggestive of tuberculosis, but the multiplicity of tubercles and atypical caseation suggested that this lesion was of sarcoid type rather than tuberculous. Therefore, the diagnosis made from biopsy of the bronchus was probable sarcoid tissue. On July 14, 1959, a right cervical lymph node biopsy was performed. The nodes as exposed were grossly abnormal and histologically showed clear cut evidence of non-caseating epitheloid-type follicles with giant cells, typical findings of sarcoidosis. In addition, it was also reported that the cultures of the bronchoscopic secretions and of three gastric washings were reported negative for tubercle bacilli by culture. The patient

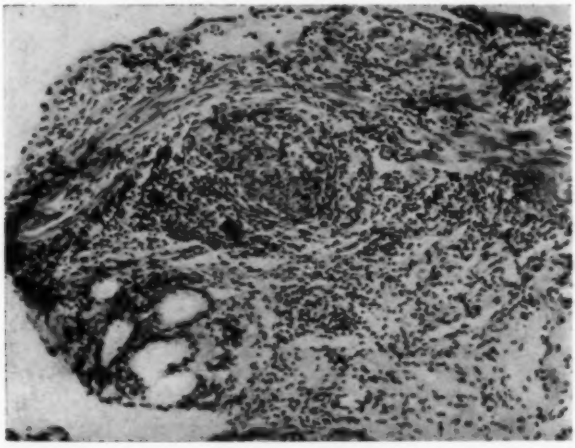

FIGURE 5: Case 2, Bronchoscopic biopsy, July 13, 1959, showing a non-caseating epitheloid follicle with giant cell.

was placed on steroid therapy (Dexamethazone, 3 mgm. daily) and given isoniazid (300 mgm. daily) for prophylaxis. Upon discharge from the hospital on July 29, 1959, the patient was asymptomatic. She was continued on the same dosage of steroids at home. A chest film on September 3, 1959 showed slight but definite generalized improvement of the diffuse pulmonary sarcoid lesions.

The 28 previously documented cases reported in the interim are shown in Table 1.

TABLE 1 — REVIEW OF PREVIOUSLY DOCUMENTED CASES

Author	Year	No. of Cases	Bronchoscopic appearance	Histology
Benedict & Castleman[1]	1941	1	Bleb-like, gray intrinsic lesions whitish fibrous stenosis	Chronic epitheloid cell granuloma non-caseating.
Olsen[5]	1946	1	nodular mucosa	Chronic granuloma Absence of caseation.
Vogt[7]	1949	1	Necropsy: Dusky red mucosa	Sarcoidosis
Jacobs[6]	1949	1	Extrinsic compression of bronchus flat, faintly hemorrhagic areas	Non-caseating tubercles.
Harvier[8]	1950	1	Thickened mucosa, Rt. bronchus	Non-caseating epitheloid cell follicles, surrounded by hyalinization.
Siltzbach, Som[9]	1952	2	Thickening and stenosis of Right middle lobe bronchus	Epitheloid cell follicles Schaumann bodies.
Turiaf *et al.*[10]	1952 1955	6	1 necropsy: Diffuse invasion of both bronchi by granulations, narrowing of main stem bronchi. 5 endoscopic: Reddened friable mucosa or lip like projection of an upper lobe margin or edematous segmental branch mucosa or slightly thickened rosy mucosal areas in Rt. main and middle lobe bronchi or diffuse reddening with multiple areas of thickening of right main and both upper lobe margins.	Various epitheloid-cell infiltrates extending into submucosa. Non-caseating granulation tissue, typical of sarcoidosis.
Cowdell[11]	1954	1	Not given	Granulation tissue typical of sarcoidosis
Grimminger[12]	1955	2	Mucosal infiltrates of carina left main and portions of right main bronchus. 40 per cent stenosis of Rt. upper lobe by conical, glassy-edematous, rose colored nodules, later seen as brownish-flat projections.	Epitheloid-cell infiltrates, no giant cells, no caseation, tendency to scar formation and sclerosis.
Citron & Scadding[13]	1957	3	Stenosing, stricturing endobronchial granulomatous lesions, some showing fibrosis.	Non-caseating epitheloid tubercles.
Kalbian[14]	1957 (11)	3	Edematous, inflamed, thickened mucosa of granular appearance. Also extrinsic compression.	Non-caseating tubercles, Sarcoidosis.
Honey & Jepson[15]	1952	2	Generalized inflammation, slit-like narrowing of upper lobe bronchus. Severe distortion and rigidity of entire bronchial tree.	Discrete epitheloid-cell follicles, giant-cells with asteroid bodies, some necrosis no caseation, some hyalinization.
Dijkstra[16]	1958	6	Edematous, reddened mucous membrane; occasionally areas of granulation or stenotic divisional branches. Sometimes severe stenosis.	Non-caseating tubercles, confirmatory of sarcoidosis.
Total documented cases exclusive of necropsies		Total: 28		

Discussion

Kalbian[14] in his discussion believes that there are three possible abnormal bronchoscopic findings, in pulmonary sarcoidosis, namely: 1. Signs of external pressure from enlarged lymph nodes; 2. Granular, nodular, rough-looking mucous membrane with small blebs; 3. Thickened, edematous mucous membrane with stenosis of the bronchus. Grimminger[12] attempts to arrive at a characteristic classification on the strength of his two cases with multiple observations. One would tend to identify his cases with Kalbian's first and second categories respectively. Turaif *et al.*[10] in their series report findings which would fit into the classification mentioned above, but some of their findings are minor and localized, similar to those in our two cases. Their positive biopsies encourage one to attempt biopsy from even a trivial variation of normal if one considers sarcoidosis as a possible etiology. Citron and Scadding[13] rightly conclude that the bronchoscopic appearance of the mucosa varies with the stage of the disease. They feel that bronchial involvement is widespread and ranges from acute changes with edema, granulation tissue and nodular areas to fibrotic, firm, smooth or irregular areas of distortions and strictures. One would be inclined to agree with the French authors[9,10] that endobronchial sarcoid infiltrations are analogous to those elsewhere in the respiratory apparatus such as nasal accessory sinuses, tonsils and larynx[2,5] and it is believed they often reflect the sarcoid changes in the lung. Their location helps to explain the presence of respiratory symptoms such as wheezing, the accompanying atelectasis that is occasionally found or frequent obstructive emphysema. Thus, these lesions tend to explain further the polymorphy of the pulmonary form of Boeck's sarcoid.

Most authors feel that when pulmonary sarcoidosis is suspected, a bronchoscopic biopsy should be attempted.[10,11,12,14] One author advocates[10] bronchoscopy and biopsy in those cases that have clinical features of recurrent bronchopulmonary infection, stridor and progressive dyspnea. Another[11] advocates a relatively large biopsy bite to provide a reasonably large portion of tissue, since a small specimen may contain insufficient granulomata to show the general uniformity of the disease pattern. The authors of this paper believe that the only logical approach is that of careful small, possibly multiple biopsies from areas suggestive of involvement. The judicious use of this method will frequently confirm or lead to a correlation with the clinical and roentgenologic diagnosis of sarcoidosis. Therefore this is considered the procedure of choice.

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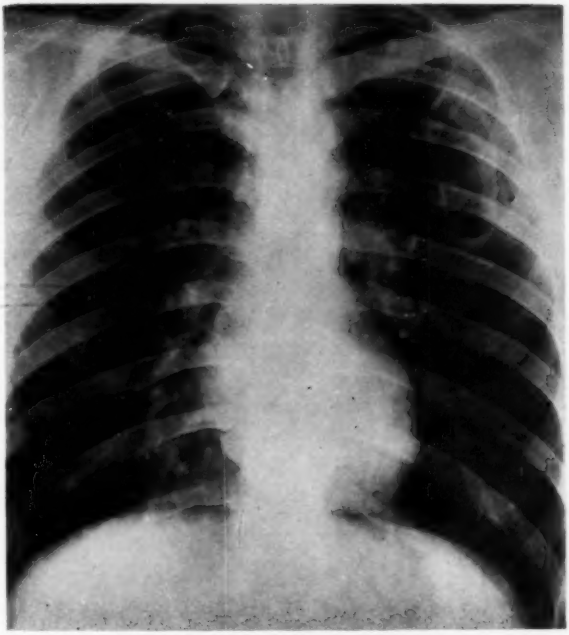
X-RAY FILM OF THE MONTH

Edited by Benjamin Felson, M.D.

Clinical History

This patient is a 39 year-old laborer from the San Joaquin Valley who presented with the symptoms of fever, malaise, arthralgia and headache. These symptoms had been present intermittently for two years and were slowly progressive. No respiratory symptoms had ever been noted. Past history revealed a childhood exposure to tuberculosis and a long history of drinking unpasteurized milk.

The chest film revealed a thin-walled cavity in the posterior segment of the left upper lobe without surrounding inflammatory disease. Repeated smears for tuberculosis, skin tests for coccidioidomycosis, histoplasmosis, and tuberculosis, and complement fixation tests for coccidioidomycosis and brucella were all negative.



Answer: COCCIDIOIDOMYCOSIS

While under observation, the cavity slowly enlarged, and a segmental resection was performed. *Coccidioides immitis* was recovered from the resected specimen.

Other pulmonary manifestations of coccidioidomycosis besides cavitation are hilar adenopathy, non-specific bronchopneumonia and the coin lesion, or coccidioma. Approximately 2 to 3 per cent of patients ill enough to seek medical attention have cavities. These may be anywhere in the lung and may develop at the site of a bronchopneumonia. They are

typically thin-walled without surrounding parenchymal inflammation, although a coccidioidal bronchopneumonia or secondary infection may coexist with the cavity, making differentiation from tuberculosis or lung abscess difficult. Indeed, several cases of coccidioidomycosis and tuberculosis in the same patient have been reported.

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The Committee on Chest Roentgenology welcomes comments. We would also be pleased to receive x-ray films of exceptional interest with brief history. Please submit material to: Benjamin Felson, M.D., chairman, Department of Radiology, Cincinnati General Hospital, Cincinnati, Ohio.

PRESENT STATUS OF BRONCHOPLASTY

Recent literature on bronchoplasty was reviewed and indications, techniques and results of 285 cases were introduced. The results of this surgical procedure have been satisfactory. It should be performed more frequently for repair of chronic airway stenosis and treatment of cancer and trauma.

Suturing, anastomosing and partial transplantation methods have yielded good results. End-to-end anastomosis of the bronchus is the best for repairing the resected bronchus, but the length of the resection is naturally limited in this way. The upper limit of the length of resection which can be anastomosed has been found by the author.

On the main bronchi, 4 cm. length of resection is anastomosable when upper lobectomy is combined.

Dotai, Y.: "Present Status of Bronchoplasty," *Lung and Heart (Japan)*, 7:315, 1960.

FIBROELASTOSIS OF THE HEART: CLINICAL AND HEMODYNAMIC FEATURES OF 18 CASES

In fibroelastosis of the heart, the symptoms of frequent respiratory infections, failure to grow, pallor and dyspnea are nonspecific. However, when these are associated with a gallop rhythm and with signs of cardiac enlargement or failure, the condition may reasonably be suspected. Supporting radiologic evidence of left atrial enlargement and normal lung vascularity and P-wave perversion on the electrocardiogram make the diagnosis much more likely.

In the older child, the differential diagnosis from the effects of rheumatic fever may be more difficult. However, if marked failure of growth and chest deformity are present in a patient with mitral valve disease, this argues that the condition has been present for some years; back-calculation to height and weight ages will frequently put the individual in an age group (for example, two to four years), in which rheumatic fever is less likely.

Maxwell, G. M., and White, D. H., Jr.: "Fibroelastosis of the Heart: Clinical and Hemodynamic Features of 18 Cases," *Med. J. Australia*, II:774, 1960.

SECTION ON CARDIOVASCULAR DISEASES

Congenital Intracardiac Defects plus Pericardial Disease: Clinical and Surgical Findings in Six Cases*

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Pericardial disease superimposed on a congenital heart lesion, if unrecognized, may simulate a complicating condition such as failure of the right side of the heart. In this situation, one might think that an inoperable lesion or one associated with an extremely high surgical risk was present, which would untowardly influence the decision as to surgical correction.

While it is rare to encounter a patient with both pericardial disease and an intracardiac defect, such a combination was diagnosed in six patients seen at the Mayo Clinic during the past five years. All had cardiac catheterization and subsequent surgical treatment for their combined cardiac lesions. When both conditions coexisted, the clinical diagnosis as well as the decision concerning operative repair became much more difficult. The experience gained from these six patients forms the basis of this report.

Clinical Data

The surgical findings in the six patients are listed in Table 1. Four of the patients had atrial septal defects, and one had anomalous venous connection of the right lung and a patent foramen ovale. The remaining patient (Case 4) had pulmonic stenosis with intact atrial and ventricular septa. Associated chronic constrictive pericarditis was present in Cases 1 and 3, and fibrinous pericarditis with a pericardial effusion of 300 ml. was present in Case 2; the other three patients (Cases 4, 5 and 6) all had coexisting large pericardial effusions.

The principal clinical manifestations in these patients are summarized in Table 2. The presenting features in the majority were related to congestive phenomena or ascites. A murmur was first heard as early as 4 years of age and as late as 18 years. Easy fatigability and dyspnea on effort were common. Precordial pain was another frequent complaint, being described usually as a type of thoracic-wall muscular discomfort that was not consistently related to effort. Orthopnea was conspicuously

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absent in five patients and equivocally present in one. In none was there a history of antecedent rheumatic fever, tuberculosis or trauma to the thorax.

In all but one instance, the venous pressure was abnormally increased as estimated clinically, and this was confirmed at the time of cardiac catheterization. Hepatomegaly was present in two thirds of the patients, and half of them had ascites. All had systolic murmurs of grade 1 to 2 (on the basis of 0 to 4), usually heard best over the left second and third intercostal spaces or near the region of the cardiac apex. Diastolic murmurs were absent in all but one patient (Case 6), in whom a faint mid-diastolic murmur was heard at the apex. The second sound in the pulmonary area was accentuated in three patients, normal in two and decreased in one; this last patient had severe pulmonic stenosis and was the only one manifesting mild peripheral cyanosis and digital clubbing. A friction rub was audible in two patients, and a "paradoxic" pulse was noted in one.

Laboratory Findings

Results of routine laboratory studies, such as hemoglobin, leukocyte and differential cell counts, sedimentation rates, urinalyses and blood urea, were essentially normal in all but one patient (Case 2), who had hypochromic anemia attributed to menorrhagia. None had secondary polycythemia. No etiologic factors could be elicited from such studies as tuberculin skin tests, serologic tests for syphilis, L.E. clot preparations, or cultures of the blood for brucella and pyogenic organisms. Cytologic examination of the pericardial fluid was negative for malignant cells. Similarly, bacteriologic studies of the excised pericardial tissues were negative for acid-fast bacilli, brucella and fungi.

Roentgenographic and Electrocardiographic Data

Generalized enlargement of the cardiac silhouette was seen uniformly in thoracic roentgenograms. The pulmonary vascular markings were increased in all but the patient with pulmonic stenosis, the latter having normal pulmonary vasculature. Decreased cardiovascular pulsations

TABLE 1—SURGICAL FINDINGS IN SIX PATIENTS WITH PERICARDIAL DISEASE AND CONGENITAL INTRACARDIAC DEFECTS

Case	Age (yr.) and sex	Findings at operation
1	17 M	ASD;* chronic constrictive pericarditis
2	31 F	Anomalous venous connection from right lung to inferior vena cava; valvular-competent patent foramen ovale; fibrinous pericarditis and pericardial effusion (300 ml.)
3	33 M	ASD;* tricuspid insufficiency; chronic constrictive pericarditis
4	41 F	Pulmonary stenosis; pericardial effusion (1200 ml.)†
5	47 F	ASD;* pericardial effusion (1000 ml.)‡
6	48 F	ASD;* pulmonary hypertension; pericardial effusion (1500 ml.)

*ASD=atrial septal defect.
†Plus preoperative pericardicentesis of 1200 ml.
‡Plus preoperative pericardicentesis of 200 ml.

were observed during fluoroscopy in three patients (Cases 2, 4 and 5). None of the patients showed roentgenologic signs of pericardial or intracardiac calcification.

Right ventricular preponderance was evident in the electrocardiograms of the six patients; in addition, two had right bundle-branch block. A suggestive clue to the presence of pericardial compression was the finding of low amplitude in the QRS complexes in two patients and borderline amplitude in a third.

Cardiac Catheterization

As noted previously, all the patients underwent cardiac catheterization. The pressure in the brachial veins and right atrium was increased in five patients and was at the upper limit of normal in one (Table 3). Four patients (Cases 1, 2, 3 and 4) had increased diastolic pressure in the right ventricle, but in only one patient (Case 1) did the contour of right ventricular pressure show the early diastolic dip followed by a diastolic plateau that is compatible with impaired diastolic filling of the ventricle. Pulmonary hypertension of varying severity was present in all except the patient with pulmonic stenosis, and in none was the systemic arterial pressure increased.

The catheter passed through an interatrial communication in three patients, and the left atrial pressure was moderately increased in three patients (Cases 1, 2 and 6).

A left-to-right shunt at the atrial level of more than 45 per cent was present in five patients and absent in the patient who had pulmonic stenosis (Table 4). Veno-arterial shunts were detected from dye-dilution curves in two patients; these shunts amounted to 5 per cent in Case 1 and 12 per cent in Case 3. Systemic arterial oxygen saturation was normal in all but the patient with pulmonic stenosis, whose oxygen saturation in the radial artery was 93 per cent. A thoracic roentgeno-

TABLE 2—PERICARDIAL DISEASE COMPLICATING CONGENITAL INTRACARDIAC DEFECTS: CLINICAL MANIFESTATIONS*

	Cases					
	1	2	3	4	5	6
Age (yr.) and sex	17 M	31 F	33 M	41 F	47 F	48 F
Murmur heard first, age (yr.)	5	18	12	4	12	15
Presenting illness	Ascites	Failure	Dyspnea	Failure	Leg pains	Failure
Dyspnea	+	+	+	+	−	+
Orthopnea	−	−	−	−	±	−
Thoracic pain	−	+	−	+	+	−
Physical findings						
Venous pressure	+3	+2	+1	+3	Normal	+3
Hepatomegaly	+	+	−	+	−	+
Ascites	+	−	−	+	−	+
Peripheral edema	−	−	−	+	−	+
Systolic murmur, grade	2	1	2	2	1	2
Second sound (P.A.)	+2	+1	Normal	−1	Normal	+2
Third sound	−	−	−	+	−	−
Friction rub	+	+	−	−	−	−
Paradoxic pulse	+	−	−	−	−	−

*Present=+; absent=−. Figures with + or − signs show numerical degree of increase or decrease.

gram taken during cardiac catheterization on this patient showed the catheter coiled in a loop in the right atrium. The right cardiac border was 2 to 3 cm. beyond the catheter lying against the right atrial wall, which was suggestive of a large pericardial effusion. This finding was also present, in retrospect, in another patient (Case 6).

Hence, from the catheterization data, the intracardiac defect was recognized in all six patients; however, in only three patients (Cases 1, 4 and 6) was there additional evidence suggesting the coexistence of pericardial disease.

Surgical Results

The only surgical death in the present group was in Case 6, in which were present severe pulmonary hypertension and a left-to-right shunt of 75 per cent at the atrial level. Ventricular fibrillation and cardiac arrest occurred suddenly during operation and failed to respond to defibrillatory measures. The other five patients underwent thoracotomy with successful alleviation of their pericardial disease and repair of their intracardiac defects. Two patients (Cases 4 and 5) with effusions had preoperative pericardicentesis, while two patients (Cases 1 and 2) had pericardial biopsy and decortication for constrictive pericarditis prior to closure of the atrial septal defects.

Postoperatively, the cardiac situation generally improved, with the exception of one patient (Case 4), who suddenly died in congestive heart failure three months after operation. The longest follow-up to date among the others was in a patient (Case 2) who was asymptomatic more than five years after the cardiac operation.

Comment

Since the clinical picture in these patients with pericardial disease resembled closely that of other patients having congestive failure consequent to congenital heart disease, further diagnostic differentiation is warranted. The latter patients with right ventricular failure generally have increased venous and right atrial pressure in conjunction with an increased pulmonary vascular resistance. Reversal of the intracardiac shunt may well occur, with considerable blood being shunted from right to left and consequent arterial oxygen desaturation and central cyanosis.

Differentiation from tricuspid insufficiency also may be difficult, but this is possible if one notes systolic expansion of the jugular pulse and intensification of the systolic murmur during inspiration,[1] and palpates a pulsatile liver. In addition, tricuspid insufficiency may be demonstrated by dye-dilution technics at cardiac catheterization.[2]

Certainly, if one suspects the coexistence of pericardial compression with congenital heart disease, surgical correction should be seriously considered, and it should be offered providing there are no contraindicating factors, such as an excessively high pulmonary vascular resistance and a predominant right-to-left shunt.

Thus, when confronted with a patient presenting the picture of congestive heart failure in association with a congenital intracardiac defect, one should consider the

TABLE 3—PERICARDIAL DISEASE COMPLICATING CONGENITAL INTRACARDIAC DEFECTS: CARDIAC-CATHETERIZATION FINDINGS

	Pressure (mm. Hg.) with patient breathing room air					
	1	2	3	4	5	6
Right atrium	27/22	26/16	17/7	30/20	13/5	23/14
Right ventricle	41/15-25*	47/20*	50/3-18	228/18-25	38/6-11	127/9
Pulmonary artery	51/31	55/30	46/20	20/13	38/12	114/39
Left atrium	26/20	27/20†	18/9	—	16/10‡	22/14
Systemic artery	106/62	116/80	123/74	106/62	128/74	101/74

*While breathing 99.6 per cent oxygen.

†Pulmonary-artery "wedge."

‡Pulmonary vein.

TABLE 4—PERICARDIAL DISEASE COMPLICATING CONGENITAL INTRACARDIAC DEFECTS: FURTHER CARDIAC-CATHETERIZATION FINDINGS

	Patient breathing room air					
	1	2	3	4	5	6
Oxygen saturation of blood, per cent						
Mixed venous*	57	75	66	58	78	65
Right atrium	85	90	87	52	96	87
Right ventricle	—	—	88	61	93	88
Pulmonary artery	77	87	89	56	93	88
Systemic artery	96	96	96	93	97	95
Blood flow, L./min./M²						
Pulmonary	3.5	9.6	9.0	1.9	18.4	8.1
Systemic	1.9	4.8	2.6	(No shunt)	4.1	2.5
L-R shunt, per cent of pulmonary flow	46	46	66	0	75	75

*Obtained from sum of oxygen saturation of the blood in superior vena cava and that in inferior vena cava divided by 2.

possibility of coexistent pericardial disease, especially if there is *high venous pressure* with little or *no right-to-left shunt* and *normal arterial blood oxygen saturation*. Even so, pericardial disease complicating congenital malformations of the heart may pose a most difficult challenge to the cardiologist as well as to the surgeon; yet, when these conditions are properly recognized and corrective operations performed, the treatment provides a most hopeful and gratifying result to both the patient and his physician.

SUMMARY AND CONCLUSIONS

Pericardial disease complicating congenital lesions of the heart is uncommon. A study has been made of six patients having this combination.

Two patients had an atrial septal defect and chronic constrictive pericarditis, and two had an atrial septal defect and pericardial effusion amounting to 1200 and 1500 ml., respectively. One had severe pulmonic stenosis and pericardial effusion of 2400 ml. The sixth patient had anomalous connection of the right pulmonary veins and fibrinous pericarditis. All the patients underwent cardiac catheterization and subsequent surgical repair, which substantiated the existence of the intracardiac and pericardial lesions.

When a patient with congenital heart disease presents clinical manifestations of congestive heart failure, one should consider the possibility of coexistent pericardial disease, especially if there is increased venous pressure with normal oxygen saturation of the arterial blood. Thus, it is important to identify the association of pericardial compression and congenital heart disease, as both are usually correctable by present-day surgical technics.

RESUMEN Y CONCLUSIONES

No es común la afección pericárdica que complica a las lesiones congénitas del corazón. Se ha hecho el estudio de seis enfermos que tienen esta combinación.

Dos enfermos tenían un defecto del tabique auricular y pericarditis crónica constrictiva y dos tenían comunicación interauricular y derrame pericárdico con líquido de 1200 a 1500 ml. respectivamente.

Uno tenía estenosis pulmonar severa y derrame pericárdico de 2,400 ml. El sexto enfermo tenía conexión anómala de las venas pulmonares derechas y pericarditis fibrinosa. Todos los enfermos se sometieron a cateterización y subsecuente reparación quirúrgica que comprobó la existencia de las lesiones intracardiacas y pericárdicas.

Cuando un enfermo con enfermedad congénita del corazón presenta manifestaciones clínicas de insuficiencia cardiaca congestiva, debe uno considerar la posibilidad de coexistencia de la enfermedad pericárdica especialmente si hay aumento de la presión venosa con saturación normal de oxígeno en la arteria pulmonar. Así, es importante identificar la asociación de compresión pericárdica y la enfermedad cardiaca congénita ya que ambas son corregibles por los recursos quirúrgico actuales.

RESUMÉ

Il est inhabituel que des lésions congénitales du coeur se compliquent d'atteinte péricardique. Une étude a été faite sur six malades atteints d'une telle association.

Deux malades avaient une communication interauriculaire et une péricardite chronique constrictive, et deux avaient une communication interauriculaire et un épanchement péricardique atteignant 1,200 et 1,500 ml. respectivement. Un malade avait une sténose pulmonaire grave et un épanchement péricardique de 2,400 ml. Le

sixième malade avait une connection anormale des veines pulmonaires droites et une péricardite fibrineuse. Tous les malades subirent un cathétérisme cardiaque et une opération chirurgicale consécutive, qui objectiva l'existence de lésions intracardiaques et péricardiques.

Quand un malade atteint d'affection cardiaque congénitale présente des manifestations cliniques de défaillance cardiaque, on devrait considérer la possibilité d'une affection péricardique coexistante, particulièrement s'il y a une augmentation de la pression veineuse avec saturation oxygénée normale du sang artériel. C'est pourquoi il est important d'identifier l'association de compression péricardique et de maladie cardiaque congénitale, puisque les deux sont habituellement corrigibles par les techniques chirurgicales actuelles.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNG

Eine Perikard-Erkrankung als Komplikation angeborener Herzveränderungen ist ungewöhnlich. Es wurde eine Untersuchung unternommen an 6 an dieser Kombination leidenden Kranken.

Zwei Kranke waren Träger eines Vorhof-Septum-Defektes und einer chronischen verengenden Perikarditis; zwei hatten einen Vorhof-Septum-Defekt und einen perikardialen Erguß im Ausmaß von 1,2-1,51. Ein Kranker hatte eine schwere Pulmonalstenose und einen perikardialen Erguß von 2,4 1. Der sechste Kranke schliesslich besaß eine anormale Einmündung der rechten Pulmonalvene und eine fibrinöse Perikaraditis. Bei allen Patienten wurde eine Herzkatheterisierung vorgenommen und die Defekte anschliessend chirurgisch beseitigt, wobei das Bestehen der intrakardialen und der perikardialen Veränderungen bestätigt wurde.

Wenn ein Kranker mit angeborener Herzkrankheit klinische Manifestationen von Herzversagen mit Stauung darbietet, so muß man die Möglichkeit einer gleichzeitig bestehenden Perikarderkrankung in Erwägung ziehen, ganz besonders dann, wenn der venöse Druck erhöht ist bei normaler Sauerstoffsättigung des arteriellen Blutes. Est es doch deshalb auch von Wichtigkeit, die Verknüpfung einer Perikard-Kompression mit angeborener Herzkrankheit zu erkennen, weil beide mit der heutigen chirurgischen Technik zu beheben sind.

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SURGICAL TREATMENT OF CARDIAC ANEURYSMS FOLLOWING MYOCARDIAL INFARCTION

Cardiac aneurysms which develop after infarction of the myocardium are mostly located in the left ventricular region. Since the successes of heart surgery and poor prospects of conservative treatment of aneurysms associated with multiple hazards are quite obvious, the indications for surgery in such cases should be extended.

We have devised three variants of the operative procedure for cardiac aneurysms: 1) diaphragmoplasty, 2) suturing through the aneurysm at its origin and with subsequent diaphragmoplasty, and 3) excision of the aneurysm with the removal of the sac and clots (also with diaphragmoplasty). These three variants might be used depending on the shape of the sac, its size and other conditions. The results of the surgery in 15 patients with cardiac aneurysms are favorable in general (one case died). This makes it possible to recommend careful surgery for this severe and up-to-now uncurable condition.

Petrovsky, B.V.: "Surgical Treatment of Cardiac Aneurysms following Myocardial Infarction," *Review of Surgery (USSR)*, 85:9, 1960.

RETROGRADE CATHETERIZATION OF THE LEFT VENTRICLE IN AORTIC STENOSIS

In 150 patients suspected of having aortic stenosis, retrograde catheterization of the left ventricle through the right brachial artery at the bend of the elbow was attempted. In patients with severe aortic stenosis, we succeeded in reaching the left ventricle in 66 per cent of the cases; this percentage rose to 77 per cent in patients who had only a slight stenosis or none at all. The examination was combined with catheterization of the right heart under the same basal conditions and with simultaneous determination of the cardiac output. Thus, the size of the functional aortic valve ostium could be measured with the aid of the Gorlin and Gorlin formula. In our series, there was no mortality; cardiac complications or disturbances of the circulation in the forearm need not be feared.

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Atrial Fibrillation*
Reversion to Normal Sinus Rhythm

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Auricular, or atrial fibrillation, is one of the most common cardiac arrhythmias. The mechanism of diagnosis will not be considered here, but rather the clinical criteria for attempted reversion to normal sinus rhythm. This report is concerned with the chronic type associated with severe organic heart or vascular disease, and in those persons previously considered bad risks.

The treatment of atrial fibrillation has been, and still is, a controversial subject. There is agreement that the ventricular rate should be controlled with digitalis. It also is agreed that the manifestations of the associated heart disease should be treated as indicated. However, the dispute arises over the question as to whether or not to allow patients to remain in atrial fibrillation. In the past, the general attitude has been conservative. Recently, some physicians have advised that no attempt at reversion be made because the fibrillation with a slow ventricular rate is practically as efficient as normal rhythm. Hecht, Osher, and Samuels[1] showed that cardiac reserve during exercise is greatly improved on reversion of auricular fibrillation to normal sinus rhythm, but that it is not greatly changed at rest. Griggs, Hadley and Stevens[2] called attention to the greater acceleration of the heart rate with moderate exertion in atrial fibrillation which decreased cardiac efficiency. Furthermore, the studies of Kory and Meneely[3] by cardiac catheterization have shown that persons with atrial fibrillation who have been compensated by all possible means except quinidine, have low resting and exercise cardiac output, but that after reversion to normal sinus rhythm the output is increased during rest and exercise.

We believe that each patient must be considered individually, but in general, every patient should have the benefit of an attempt to return the atrial fibrillation to a normal sinus rhythm. Those opposed to this idea advance the argument that there is a great danger of embolization with reversion as the atria are likely to detach portions of intramural thrombi with the first forcible contractions. We feel that this danger is more apparent than real because we have had many more episodes of embolization occur in patients who remained in atrial fibrillation.[4-8] Furthermore, we cannot agree with the statement that atrial fibrillation with a slow ventricular rate is just as efficient as normal sinus rhythm, because congestive heart failure occurs earlier and more frequently in the former.

It has been stated that no attempt to induce reversion should be made if atrial fibrillation has been present longer than a certain period time. This varies anywhere from two weeks to two years depending upon the authority quoted. We believe that the time interval is not

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important. However, the longer the duration of an arrhythmia the more difficult it is to revert to and maintain a normal rhythm. There are many individual exceptions to this, but it applies to the average case. It has also been stated that any patient who has ever had an embolic episode should not be reverted because he is much more likely to have another embolus during the process. We do not agree with this statement. We have found that a patient who has had an embolus while in atrial fibrillation is definitely subjected to recurrent emboli if the atrial fibrillation continues. This risk is much greater than that of an embolism during reversion. Goldman[9] concluded that the chances are seven to one in favor of a patient suffering an embolism while in atrial fibrillation as compared with the likelihood of occurrence at the time of reversion to normal sinus rhythm. In general, we would state that there are no absolute contraindications to return of atrial fibrillation to normal sinus rhythm.

In 1953, 368 cases of atrial fibrillation were collected and tabulated from the histories of 8000 consecutive cardiovascular patients.[10] Of these, 298 (81 per cent) were returned to normal sinus rhythm without a fatality, but with cerebral embolism resulting in aphasia in two cases. (Table 1)

TABLE 1

Total No. of Cases.	368	
No. of cases converted.	298	81 per cent
Embolism subsequent to conversion.	2	0.5 per cent
Fatalities attributed to therapy.	0	0.0 per cent

Thrity-eight of the patients were returned to normal sinus rhythm more than once, and one was returned from atrial fibrillation on 17 different occasions. This patient had rheumatic heart disease with mitral stenosis and insufficiency and at the time of the reversion was one of the two that developed aphasia, probably secondary to a cerebral embolus.

In this series, the largest dose of quinidine employed was given to a physician who was converted from atrial flutter to fibrillation, and then required 1000 grains (65 gm.) given in one week before he returned to sinus rhythm. In normal rhythm, this patient experienced headaches that he could not tolerate and stopped the quinidine. He immediately returned to atrial fibrillation and had a fatal pulmonary embolus nine months later.

In the severe cases that did not revert to normal rhythm on a high dose of quinidine, we found that if we stopped the drug entirely for a day or two and then resumed the previous maximum dose, many reverted.

In eight cases, atrial flutter was converted to fibrillation by digitalis before reversion to normal sinus rhythm with quinidine. In our opinion, it was more difficult to change the atrial flutter to fibrillation than to change the fibrillation to normal sinus rhythm. Several patients changed from atrial fibrillation to a nodal rhythm. In all but two cases, this was merely a transient phase prior to the development of normal sinus rhythm. The two exceptions remained in nodal rhythm permanently. One patient converted to nodal rhythm which was considered permanent,

had a normal sinus rhythm two years later. Another patient developed 2:1 A-V block and later changed to delayed A-V conduction which persisted.

The observation made on this first series of cases treated between 1925 and 1945 impressed upon us our timidity in the choice of patients for the attempt to induce reversion. We realized that as years went on, we had gradually selected more poor risk patients. To reappraise our impressions, we reviewed 600 cardiac cases observed between 1945 and 1950. There were 45 patients or 7.5 per cent with atrial fibrillation. In this second series, 80 per cent of the attempts to induce reversion were successful. (Table 2) Six patients returned to normal sinus rhythm on digitalis alone. Three patients failed to change under moderate dose of quinidine and there were two cases of atrial flutter that were converted to atrial fibrillation prior to reversion to normal sinus rhythm. No embolus occurred in the patients receiving quinidine. One embolus occurred in a patient who, after having atrial fibrillation for less than 24 hours, changed spontaneously to normal sinus rhythm. Eleven patients who were not treated with quinidine had emboli while in atrial fibrillation, and five of these were later returned to normal sinus rhythm. Ten critically ill patients, five with acute myocardial infarctions, were returned to normal sinus rhythm.

TABLE 2

Total No. of Cases.	45	
No. Cases Converted	36	80 per cent
Embolism subsequent to conversion.	1*	
Fatalities attributed to therapy.	0	0.0 per cent

*Spontaneous reversion without therapy to normal sinus rhythm.

The experience obtained from the analysis of these two series of cases stimulated us in 1950 to try to revert all cases of atrial fibrillation. Between the years of 1950 and 1957, we selected from 146 cases of auricular fibrillation 93 cases that were considered a poor risk. This group showed a higher percentage of reversion, etc. than the first two series. (Table 3) We were now only concerned with obtaining experience in regard to the poor risk patients with atrial fibrillation. They were more than 65 years of age, with a few exceptions, and those with congestive heart failure, coronary occlusion with infarction, long standing fibrillation, cerebral vascular accidents, pulmonary emboli, or who had enlarged hearts. (Table 4) Three of the patients died during the attempt to change to normal sinus rhythm. They were women and two had had arteriosclerotic and hypertensive heart disease with atrial fibrillation for a number of years. The first patient was given 15 grains (1 gm.) of quinidine the first day of therapy and 20 grains (1.65 gm.) the next day when she had a fatal syncope. No autopsy was obtained, but it is our opinion that she died as the result of the attempt to induce reversion to normal sinus rhythm. The other patient died about a half hour after reversion to normal sinus rhythm and about five minutes after electrocardiographic confirmation. We were of the opinion that she had died of a pulmonary embolus. However, the autopsy revealed that she had had a thrombosis of a pulmonary artery as the result of a metastasis

from a uterine adenocarcinoma that had been resected five years previously. The third, a 32 year-old woman with rheumatic heart disease, mitral stenosis and insufficiency, developed acute pulmonary edema with the onset of atrial fibrillation. She was returned to normal sinus rhythm on the tenth day after initially receiving 20 grains of quinidine sulfate with an increase of 5 grains per day. Six months later, she discontinued her maintenance dose of 2 grains every six hours for five days and again developed atrial fibrillation. She was again started on quinidine sulfate, this time 30 grains the first day and increased by 5 grains per day. She was found dead in bed on the ninth day after receiving 70 grains of quinidine on the preceding day. No autopsy was obtained.

TABLE 3

Total No. of Cases.	93	
No. of cases converted.	83	89 per cent
Embolism subsequent to conversion.	0	0.0 per cent
Fatalities attributed to therapy.	3	3.0 per cent

TABLE 4

Total Number of cases of Auricular fibrillation	146
Cases selected as poor risks	93
Cases not returned to normal sinus rhythm (3 died)	3
Cases unable to revert	6
Auricular flutter	1
Cases of Induced Reversion	
Posterior commissurotomy	1
Pulmonary emboli	3
Cardiovascular accidents	7
Embolism of the femoral artery	2
Congestive heart failure treated, then reversion induced	20
Congestive heart failure not treated, reversion induced first	14
Pulmonary edema	2
Cases past 70 years of age	13
Cases past 80 years of age	7
Auricular flutter	2
Cases of reversion twice induced, but 3rd attempt refused	3
Post-commissurotomy reversion induced, atrial fibrillation returned, further attempt of induced reversion refused	1
Miscellaneous	8
Total	83

There were three patients who had been returned to normal sinus rhythm twice, but on developing atrial fibrillation the third time, two of them refused quinidine because of the distressing symptoms associated with the necessary large doses of the drug. The third refused because of syncopal attacks experienced during the two previous regimens and he felt that the third attempt might be fatal. There were two in their 30's with rheumatic heart disease and mitral stenosis who, after commissurotomy, went into atrial fibrillation with congestive heart failure. The congestive heart failure was always immediately associated with the onset of atrial fibrillation. Both had been returned to normal sinus rhythm twice during the past two years. One of them went into atrial fibrillation the third time, and after massive doses of quinidine

for a period of three weeks with a daily dose as high as 110 grains (7.33 gm.), refused any further attempts because of the toxic effects. He remained in fibrillation and congestive heart failure. The other patient is in normal sinus rhythm. There were six that we could not return with quinidine using all of the means at our command. All of them were given a rest after the first attempt, and a second prolonged attempt was made a month later without success.

On the brighter side, there were three who previously had experienced pulmonary emboli, and seven who had had cerebral vascular accidents all of whom were returned to normal sinus rhythm. Two had had an embolism into the femoral artery while being prepared in the hospital for reversion to normal sinus rhythm. They were successfully operated upon and later returned to normal sinus rhythm. There were 20 persons in congestive heart failure who were successfully treated before they were returned to normal sinus rhythm. There were 14 cases of slow ventricular rates and congestive heart failure in which the treatment of the atrial fibrillation was started immediately. On reverting to normal sinus rhythm, the congestive heart failure disappeared. We did not experience a high incidence of quinidine toxicity in congestive heart failure, nor did we find it necessary to treat the congestive failure first as has been reported by others.[11] There were two who developed pulmonary edema when they started into atrial fibrillation. Both returned to normal rhythm.

There was one woman, 68 years of age, who had been in atrial fibrillation since a subtotal thyroidectomy done 28 years previously. She was returned to normal sinus rhythm on 65 grains (4.33 gm.) of quinidine given over a course of three days. In this group of patients returned to normal sinus rhythm, there were 13 past 70 years of age and seven were past 80. There was one with syphilitic heart disease, aortic insufficiency and left ventricular failure who had been in atrial fibrillation for six years. Three years ago he was returned to normal sinus rhythm with no subsequent interruptions or signs of failure.

There were three persons with long-standing atrial flutter who could not be converted to atrial fibrillation by large doses of digitalis alone, but in addition, required calcium gluconate intravenously to intensify the digitalis action. Only by this method could we lower the atrial rate below 200. Afterwards, two were returned to normal sinus rhythm with quinidine, and the third, after six unsuccessful attempts, converted to atrial flutter.

A few patients had to be returned more than once. One, a hypertensive woman who is easily reverted with 50 to 60 grains (4.0 gm.) of quinidine over a two to three days period, has had to be returned about every four to six weeks over the past year. This is only because she does not have the mental capacity to follow directions. The other, a man with rheumatic heart disease and multiple valvular leesions, had been in atrial fibrillation for four years before he developed congestive heart failure. On return to normal sinus rhythm, the congestive heart failure immediately disappeared. However, since then induction of reversion has had to be repeated on three occasions.

SUMMARY

All patients in auricular fibrillation should be given an opportunity of reversion to normal sinus rhythm. The failures as in the selected cases will be about 20 per cent and the mortality 1 or 2 per cent. This is greatly outweighed by the previously stated advantages. We believe that attempts to induce reversion should be abandoned when quinidine produces severe toxic effects, especially in the large doses. The attempt should also be abandoned when there is evidence of complete heart block and ventricular arrhythmias with syncope.

RESUMEN

Debe darse una oportunidad a todos los enfermos que tenga fibrilación auricular para que se normalicen con ritmo sinusal.

Los fracasos en los casos escogidos serán aproximadamente de 20 por ciento y la mortalidad de 1 a 2 por ciento.

Esto está grandemente compensado por las grandes ventajas señaladas. Creemos que los intentos para lograr la reversión, deben abandonarse cuando la quinidina produce efectos tóxicos severos, especialmente a grandes dosis. También debe abandonarse el intento cuando es evidente un bloqueo cardiaco y la arritmia ventricular con síncope.

RESUMÉ

Tous les malades atteints de fibrillation auriculaire devraient avoir la possibilité de retrouver un rythme sinusal normal. Les échecs, comme dans les cas choisis par les auteurs, seront de l'ordre de 20% et la mortalité de 1 à 2%. Ces inconvénients sont largement compensés par les avantages constatés. Les auteurs croient que les essais pour provoquer la réversibilité devraient être abandonnés quand la quinidine provoque des effets toxiques graves, particulièrement à fortes doses. L' essai devrait être également abandonné lorsqu'il y a la preuve d'un bloc cardiaque complet et des troubles du rythme ventriculaire avec syncope.

ZUSAMMENFASSUNG

Allen Kranken mit Vorhofflimmern sollte die Möglichkeit gegeben werden zur Wiederherstellung eines normalen Sinusrhytmus. Die Versager, so wie in den ausgewählten Fällen werden etwa bei 20% liegen und die Mortalität bei 1 oder 2%. Dies wird größtenteils wieder wettgemacht durch die oben festgestellten Vorteile. Nach unserer Meinung sollte man dann die Versuche, eine Reversion zu erreichen, wieder aufgeben, wenn das Cinidin zu schweren toxischen Nebenwirkungen geführt hat, besonders in den hohen Dosen. Der Versuch muß ferner dann eingestellt werden, wenn es sich herausstellt, daß ein kompletter Herzblock besteht und ventrikuläre Arrhythmien mit Bewußtlosigkeit.

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Electrocardiographic Ischemic Patterns Without Coronary Artery Disease*

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ST segment deviation is commonly accepted as a sign of myocardial ischemia. Whether indicative of actual myocardial ischemia or not, ST segment changes have been and continue to be generally interpreted as "ischemic ST deviation," "subendocardial injury," "coronary insufficiency," or even "myocardial infarction." The use of any of these terms intimates coronary artery disease and tends to leave one with an ominous feeling about the patient's prognosis. Such ominous prognostic feelings on the part of the clinician may lead him to recommend drastic changes in the patient's way of living. Such recommendations either directly or indirectly indicate to the patient that from that time forward his life is in jeopardy, and he must be extremely careful. Whether or not such drastic steps are necessary following all cases of actual proved coronary artery disease is not a point to be considered here, but it is obvious that such total-life modifying recommendations are contraindicated when there is no coronary artery disease. Iatrogenic heart disease is disabling to a patient, and is unnecessary if the clinician clearly understands when electrocardiographic changes are significant and when they are not.

"Ischemic patterns" refers to those ST segment changes (elevation and depression) which have been commonly considered to be the result of myocardial ischemia. These same electrocardiographic changes *are* encountered in a wide variety of clinical conditions *without other evidence* of coronary artery disease. "Non-ischemic" electrocardiographic changes have received sporadic attention, but little effort has been made to establish a *common denominator* to explain these ST changes. This paper represents an attempt in this direction.

Definitive understanding of the nature of ST deviation has been hampered, both clinically and experimentally by the relative lack of concern as to whether the direction of the shift was upward or downward. It has been generally assumed that epicardial ST segment depression is reciprocal to ST segment elevation on the subendocardial layers of the wall of the heart; elevation or depression in a given lead being dependent on the point of observation, i.e., on the position of the electrode with regard to the area of myocardial injury.

Recently published experiments[1] demonstrate that ST segment depression is a manifestation of primary epicardial change, as is ST segment elevation. Primary ST segment depression over the epicardial surface of the heart has been experimentally produced without ischemia by per-

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fusion of the coronary artery with specific concentrations of electrolytes[2] sufficient to alter the extracellular concentration.

In these perfusion experiments, *no myocardial ischemia was produced*, yet both ST segment elevation and ST segment depression were recorded from the same perfused area, the direction of the deviation depending on the electrolyte composition of the perfusate.[2] Perfusion of coronary arteries with solutions of high potassium (4 m.Eq./L.K+) or low sodium (103 m.Eq./L.Na+) concentration resulted in ST segment elevation. Perfusion with solutions of high sodium (171 m.Eq./L.Na+) or low potassium (0.5 m.Eq./L.K+) concentration led to ST segment depression. (Figures 1, 2, 3, 4). These ST changes, whether produced by changes in sodium or potassium concentrations, tended to disappear shortly after termination of perfusion.

It is a well established concept that the shape of the electrocardiographic curve appears to be related to the pattern of electrolyte distribution on both sides of the cell membrane,[3-11] and that ST segment deviation reflects an alteration in these electrolyte concentrations or relationships.

Normally the ratio of intracellular to extracellular potassium is about 30:1 while sodium has an extracellular to intracellular ratio of about 10:1. It is this transmembrane ratio or gradient which has been temporarily altered in the previously mentioned perfusion experiments.[2]

Either an extracellular increase in sodium or an extracellular decrease in potassium leads to an *increased* transmembrane gradient. The increased transmembrane gradient of either of these ions is reflected in the electrocardiogram as *ST segment depression*. If the transmembrane gradient of either sodium or potassium is decreased, *ST segment elevation* will be seen.

Clinically, ST segment is usually associated with lowering of the T wave while ST elevation is usually associated with elevation of the T wave.

Clinically, alterations in the electrolyte pattern, similar to those produced in perfusion experiments have been found in two different types

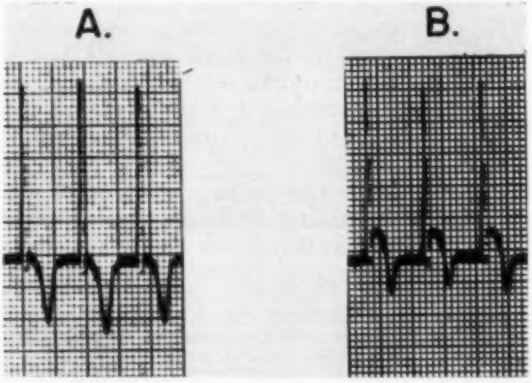

FIGURE 1: ST segment response to perfusion of high concentration potassium solution (4 m.Eq./L.K+) in saline solution (142 m.Eq./L.Na+) into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment elevation occurs with perfusion.

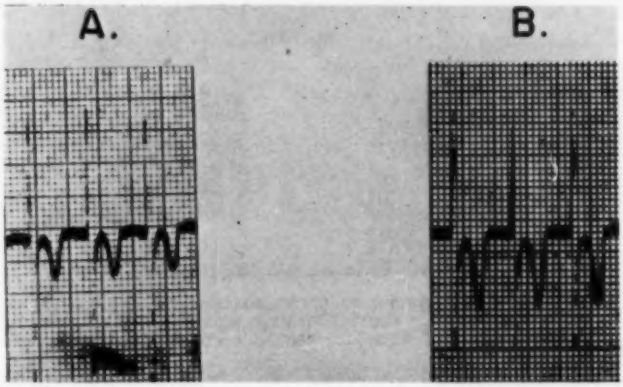

FIGURE 2: ST segment response to perfusion of low concentration potassium solution (0.5 m.Eq./L.K+) in saline solution (142 m.Eq./L.Na+) into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment depression occurs with perfusion.

of myocardial ischemia: ischemia with ST segment depression and ischemia with ST segment elevation.[2] Just as in the perfusion experiments, correlation of chemical determinations with electrocardiographic changes showed that ischemia with ST segment depression is related to an increase in the transmembrane gradient of sodium and potassium ions. In ischemia with ST elevation, there is a decrease in the transmembrane gradients, with the ischemic cell losing potassium to the extracellular compartment while the intracellular content of sodium probably increases.[2]

The described perfusion experiments are of more than theoretical interest. ST segment deviations due to electrolyte changes have been reported in many non-ischemic clinical conditions.[12,13] It is important to note that non-ischemic disease entities which are dissimilar in etiology and clinical picture often show similar changes in electrical potential, leading to identical ST segment deviations. The production of electro-

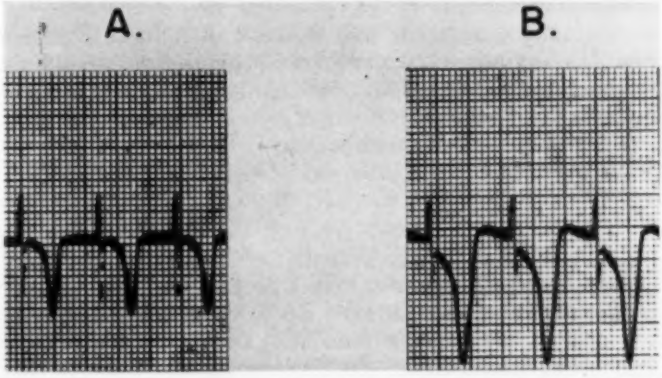

FIGURE 3: ST segment response to perfusion of hypertonic saline solution (171 m.Eq./L.Na+) without potassium into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment depression occurs with perfusion.

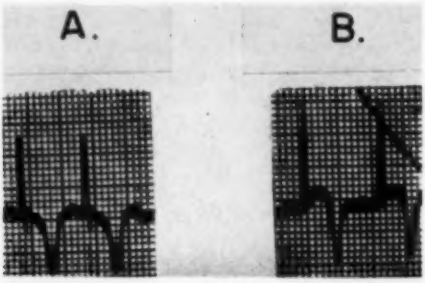

FIGURE 4: ST segment response to perfusion of hypotonic saline solution (103 m.Eq./L.Na+ with 2 m.Eq./L.K+) into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment elevation occurs with perfusion.

cardiographic changes by alterations in electrolyte balance in clinical conditions has up to now been limited largely to the obvious cases of potassium balance. However, in the perfusion experiments it has been shown that changes in sodium concentration account for ST segment deviation also. The concept of primary ST segment depression enables us to correlate the direction of ST segment shift with specific electrolyte patterns. In animal experiments and clinical conditions the direction of the ST segment response is usually the same whether on an ischemic or obviously non-ischemic basis.

It should be noted that in the perfusion experiments, the injection of solutions containing various ionic concentrations altered the extracellular fluid concentration of the heart *only* in that area supplied by the coronary artery being perfused. The ST segment deviations were observed a few seconds following the injections. Many different clinical conditions also reveal variations in serum ionic concentrations. These variations, of course, affect the ionic concentration of extracellular fluid of the *entire* heart. They may also affect the ionic concentration of the intracellular fluid. Also, the alterations of serum ionic concentration seen in these clinical conditions do not occur immediately, as they do in the perfusion experiments. The duration of the imbalance is infinitely greater in the clinical conditions than in the experiments.

There are many differences that make it difficult to compare acute experiments in animals with chronic or subacute disease states in man. However, similar electrocardiographic changes and alterations in extracellular fluid ionic concentrations are seen both clinically and experimentally. Such similar findings lead us to speculate regarding the correlations between experimental evidence and the clinical conditions described in this paper.

The speculations in this paper can only be considered preliminary in view of the insufficient data now available. It will be many more years, even decades, before all of the factors that affect ST segment deviation are known and understood. Simple acute experiments, such as these cited in this paper, are valuable, since they do help explain the "abnormal" electrocardiogram. Perhaps the most important point of this entire paper is the evidence that is presented showing that an "ischemic" electrocardiogram does not necessarily mean an ischemic heart.

In the remainder of this paper some of the non-ischemic clinical conditions showing ST segment deviation will be reviewed and discussed. For purposes of presentation these clinical conditions have been separated into four general groups, divided according to their commonly accepted etiology:

(1) electrolyte imbalance
(2) hormonal influence
(3) hemoglobin deficit or blockage
(4) enzyme system blockage

Group 1: Electrolyte Imbalance

The mechanism responsible for electrocardiographic alterations evoked by abnormal electrolyte distributions is already understood to a certain degree.[14,15] A decrease in the transmembrane gradient of cations is probably responsible for low membrane potential of the cell and for the elevation of the ST segment. An increase in the transmembrane gradient of cations,[2] on the other hand, is related to an increase in membrane potential, the latter showing ST segment depression.[2]

The ST segment responses to various *experimentally induced* electrolyte imbalances are identical in pattern to those encountered in a number of *clinical* conditions which are known to involve the same electrolyte imbalances. Under clinical conditions, however, many other factors are present, making the picture more complex. Nevertheless, it has been stated that the electrocardiogram is more sensitive than any chemical method in reflecting the seriousness of an electrolyte imbalance.[16] It would seem that this is not always true.

Electrolyte imbalance associated with ST segment depression.

Diabetic coma and hyperglycemia: In diabetic coma, important electrolyte changes are seen both before and during specific treatment. Before treatment is initiated, the potassium stores of the body are markedly depleted,[17,18] but this is masked by dehydration and the true deficit is rarely reflected in the electrocardiogram. During treatment with insulin and sodium salt infusions, the serum sodium level rises and the previously masked depletion of potassium stores becomes manifest through rehydration. This deficit is reflected in a low serum potassium level.[19,20] Serum potassium is further depressed as potassium accompanies glucose into the cell under the influence of insulin.[21] Both the high sodium and low potassium concentrations in the extracellular fluid contribute to depression of the ST segment.[2] With the infusion of appropriate amounts of potassium salts the ST segment depression will disappear.[97]

Similarly, ST segment depression occurs after injection of hypertonic glucose or the ingestion of huge quantities of glucose.[22,23] The endogenously released insulin will promote the movement of glucose and potassium into the cells, and the decreased extracellular potassium will be reflected electrocardiographically as ST segment depression.

Familial periodic paralysis: During attacks of familial periodic paralysis extracellular potassium probably migrates into the cells, resulting in a higher transmembrane gradient of this ion.[24,25,26] ST segment depression is often observed during these attacks.

Artificial kidney: The changing electrocardiographic picture during extracorporeal hemodialysis[27,29,39] from ST segment elevation to an isoelectric line, or even ST segment depression, follows closely the changing serum level of potassium. The low serum potassium level after hemodialysis usually shows ST segment depression along with other electrocardiographic changes.

Gastrointestinal conditions: ST segment depression has been observed in the following conditions: therapeutic peritoneal lavage, intestinal lavage, prolonged diarrhea,[30] in the course of severe malnutrition,[31,32] dysentery, intestinal obstruction,[33] bile fistula, and prolonged vomiting. All of these conditions are characterized by potassium loss and a low extracellular concentration of potassium. In these gastrointestinal states ST segment depression may occur without any indication of myocardial ischemia.

Salicylate toxicity: A toxic dose of salicylates directly stimulates the respiratory center, resulting in hyperventilation, loss of carbon dioxide and respiratory alkalosis. In salicylate toxicity the stage of respiratory alkalosis is associated with low serum potassium and occasionally visible ST segment depression. Marked ST depression is sometimes found.[34,36] Treatment with potassium salts has been found to return the depressed ST segment to the isoelectric line.[35]

There is evidence for the assumption that alkalosis potentiates electrocardiographic changes induced by hypopotassemia, while acidosis obscures the hypopotassemic pattern.[14,15]

Anxiety: ST segment depression has been encountered in acute anxiety reactions and is commonly ascribed to respiratory alkalosis secondary to hyperventilation.[36,37] In clinical practice, false positive exercise tests may be brought on by incidental anxiety in the emotionally labile patient.

Diuretic therapy: Cohen[38] recently described a case of muscle paralysis due to hypokalemia which had developed during the course of chlorothiazide administration. In this case ST segment depression was recorded while the serum potassium level was as low as 1.65 m.Eq./L. The serum sodium level was elevated. Hypokalemia is noted in about 40 per cent of patients during chlorothiazide treatment. ST segment depression has also been observed during treatment with mercurial diuretics.[39,40]

Low sodium diet: A very restricted low sodium diet used in the treatment of arterial hypertension has occasionally led to disappearance of the ST segment depression connected with left heart strain. Simultaneously, a substantial decrease in serum sodium and a corresponding increase in serum potassium was reported.[41] This observation raised the question as to whether the electrolyte changes secondary to severe sodium restriction were alone responsible for the electrocardiographic phenomena.[42] Using potassium in the treatment of patients with left heart strain gradually returned the electrocardiographic changes characteristic of hypertensive disease to normal. Since norepinephrine and mineralocorticoids show a specific ability to promote a shift of sodium into the cell, such electrolyte changes may be involved in the production of arterial hypertension.

Electrolyte imbalance associated with ST segment elevation.

Shock: The complex and changing mechanisms of profound traumatic shock may result in either elevation or depression of the ST segment. Usually, extracellular potassium is elevated while extracellular sodium is low.[42] ST segment elevation was found in electrocardiograms taken following war injuries, and in acute pulmonary embolism with shock.[44] Experimental ischemic compression of an extremity has led to ST segment elevation,[45] the elevation becoming more pronounced after massage of the ischemic areas. This phenomenon was explained on the basis of potassium being released from the ischemic tissues. ST segment depression, in contrast, was observed in post-operative patients who were maintained on intravenous feeding; this ST segment depression was corrected by the administration of potassium chloride.[46]

Miscellaneous conditions: Elevation of the ST segment may be encountered in clinical entities characterized by increased levels of extracellular potassium; as in some types of acidosis, following ingestion of excessive potassium salts,[47] and in uremia. In this last condition elevation of the ST segment may mask the electrocardiographic picture of "left heart strain."[48,49] High levels of serum potassium may also be observed when there is an excessive release of cellular potassium, as in hemolytic transfusion reactions,[50] hemolytic anemias, crush syndrome,[51] severe attacks of malaria,[52] starvation, and various other conditions associated with cell destruction. Wener *et al.*[50] encountered hyperkalemia and ST elevation after injection of hemolyzed red blood cells during animal experiments.

Group 2. Hormonal Influence

It is suggested that ST segment deviation induced by some hormonal factors can be explained on the basis of changes in electrolyte distribution. In different hormonal syndromes the ST segment may deviate up or down, depending on the serum concentrations of sodium and potassium. Some observers have succeeded in correcting such ST segment deviations by administration of appropriate electrolytes.

Adrenal cortical hypofunction: In severe acute adrenal insufficiency, ST segment elevation is encountered, usually combined with a high, peaked T wave; these changes disappearing after the serum potassium is returned to normal levels. In adrenal insufficiency urinary excretion of sodium is increased while excretion of potassium is decreased.[53] There is an associated loss of water with resultant depletion of extracellular fluid, reduction of plasma volume, and dehydration.[54] Fluid loss is further aggravated by migration of water into cells. Robertson *et al.*[55] found a concomitant decrease of intracellular sodium in adrenal insufficiency.

Adrenal cortical hyperfunction: In contrast to the ST segment response observed in adrenal insufficiency, administration of large doses of ACTH or adrenal cortical steroids over a long period of time may result in ST segment depression.[56,57] Such ST segment deviation was returned to the isoelectric line following administration of potassium salts.[58] ST segment depression induced by treatment with desoxycorticosterone acetate was found to be potentiated by 1 per cent sodium chloride,[59] but after administration of potassium chloride the changes were quickly reversed and the ST segment returned to the isoelectric level.[58]

Although the alterations in potassium are largely secondary to those of sodium metabolism, it is believed that the adrenal cortex exercises some specific effect on potassium metabolism. Cortisone in doses above 200 mg. per day increases urinary excretion of potassium, lowers the serum potassium, and results in salt retention. These changes lead occasionally to hypopotassemic hypochloremic alkalosis and signs of hypokalemic "toxicity." The same direction of electrolyte shift is induced by continued administration of large doses of corticosterone, desoxycorticosterone, and ACTH.[59]

Attention has been drawn recently to the effect of aldosterone, a hormone which has a sodium retention power 500 times that of hydrocortisone. ST segment depression is considered to be a diagnostic feature of aldosteronism and has been traced to hypopotassemia.[60] Examples of secondary aldosteronism can be encountered in nephrosis, congestive heart failure, hepatic cirrhosis, eclampsia, and idiopathic edema.[61] In tracings from patients with hepatic cirrhosis and hepatic coma ST segment depression has been observed.[4]

ST segment depression has been observed in some cases of Cushing's disease. Increased formation of adrenal steroids with salt retaining effects leads frequently to reduction of serum potassium and elevation of serum sodium. In a case of Cushing's disease Teabeaut restored the ST segment to the isoelectric level by potassium infusion.[62]

Epinephrine: Infusion of *large* doses of epinephrine has been reported to produce ST segment depression under experimental conditions.[63] This response may be explained on the basis of electrolyte changes in cardiac muscle. With infusion of a large amount of epinephrine, Robertson *et al.*[64] observed an initial rise in serum potassium followed within a few minutes by a steady decrease; at the same time there was an increase in serum sodium and glucose.

The ST segment depression encountered in anxiety states has been attributed by some authors to the action of released epinephrine.[38] An increase in the depth of ST segment depression was demonstrated in some emotionally labile patients after the injection of small doses of epinephrine. In normal subjects, small doses of epinephrine have no effect on the ST segment.[65]

Toxemia of pregnancy: As previously noted, epinephrine infusion may produce ST segment depression.[63] In normal pregnancy, epinephrine and other vasoconstrictor amines are inactivated by monoamino oxidase present in the placenta and the decidua. The activity of this enzyme, however, may be dependent on the oxygen tension within the placenta. In toxemia of pregnancy, placental ischemia has been demonstrated and these ischemic changes are believed to be responsible for a substantial lowering of oxygen tension and a significant decrease in the activity of monoamino oxidase.

This factor may at least partially explain the ST segment depression sometimes observed in toxemia of pregnancy.

Sex hormones: While there is some clinical evidence that the gonadal hormones affect the electrolyte balance of body fluids, this area is in need of more extensive investigation. ST segment depression has been reported

in menopause and was found to disappear with estrogen treatment[66] although the published changes do not appear to be great.

Insulin: Under the influence of insulin, both glucose and potassium leave the extracellular fluid and enter the cell. As the transmembrane gradient of potassium increases, ST segment depression appears. This electrocardiographic finding has been long observed in insulin shock in diabetes and in insulin coma treatment for psychiatric disorders.[67] In insulin-induced hypoglycemia Parrish[68] and Judson *et al.*[69] have correlated ST segment depression with the changes in serum glucose and potassium.

Group 3: Hemoglobin Deficit or Blockage

ST segment depression is a frequent finding in a group of clinical entities characterized by a deficit in active hemoglobin. The deficit may be due either to a decrease in total body hemoglobin or to a blockage of its physiological activity. In both instances, there is an identical result, *i.e.*, an insufficient supply of oxygen to the cell.

Hemoglobin deficit

Hypoxia impairs aerobic metabolism of glucose and the cell compensates by increasing its anaerobic glucose turnover. This less economical latter process requires considerably more glucose, and greater quantities of glucose and potassium enter the cell.[21,70] There is a concomitant compensatory shift of sodium to the extracellular fluid. The resultant changes in myocardial cell transmembrane gradients of sodium and potassium seem to be manifest in the electrocardiogram as ST segment depression.[2]

Anemia: Most cases of mild anemia present no electrocardiographic abnormalities because the hemoglobin deficit is compensated for by an increased rate of blood flow. In severe anemia, regardless of type, the state of the myocardial cell is affected and ST segment depression is often observed.[71-74] The same electrocardiographic finding has been reported after acute hemorrhage.[75]

In severe degrees of anemia, instances of anginal pain without overt coronary disease have been reported.[76-78] Relief of the anemia resulted in the simultaneous disappearance of both angina and the electrocardiographic abnormalities. It is important to note that anemia produced anginal pain and ST segment depression identical to that caused by ischemia. The common cause for the changes in both conditions is probably a similar disturbance of cell metabolism. It would seem that the manifestations of ischemia should not be regarded as specific.

Hemoglobin blockage

Blockage of hemoglobin activity, or the presence of abnormal hemoglobins, may change the carbohydrate metabolism of the cell in the same way as in absolute hemoglobin deficit.

Such *functional deficits* of hemoglobin occur in the presence of carboxyhemoglobin, methemoglobin, and sulfhemoglobin. There are numerous reports[79,80] of ST segment depression in the presence of these abnormal hemoglobins and the electrocardiographic changes can be uniformly explained by hemoglobin blockage. Occasional ST segment elevation is

observed with carbon monoxide poisoning and this may be due to an extreme degree of hemoglobin blockage.[81] The reversal of direction in the ST segment response resembles the difference observed in varying degrees of ischemia, *i.e.*, ST segment depression with mild ischemia and ST segment elevation with severe ischemia.

An insufficient oxygen supply to the alveolar surfaces of the lungs will decrease the quantity of available oxyhemoglobin and increase the amount of circulating reduced hemoglobin. This occurs in chronic pulmonary diseases, in some normal subjects during anoxemic tests, and in the presence of low atmospheric pressures at high altitudes. ST segment depression in these conditions has been extensively documented and reported.[82,83] In the final stage of anoxia, ST segment elevation may occur.

Insufficient ventilation during anesthesia results in an accumulation of carbon dioxide which combines with hemoglobin, thus reducing the amount of hemoglobin available for oxygen transport. This strong affinity of hemoglobin for carbon dioxide is more apparent in carbon dioxide poisoning which may lead to ST segment depression or to ST segment elevation in severe cases. Altschule and Sulzbach[85] showed that inhalation of 5 per cent carbon dioxide and 95 per cent oxygen results in marked ST segment shifts from standard leads. These shifts were rapidly reversible upon stopping the inhalation. Their important conclusion was that such ST segment changes occur in the absence of anoxia or ischemia.

Group 4: Enzyme System Blockage

The correlation of electrocardiographic changes with alterations in complex enzymatic reactions, with present knowledge, is largely based on reasoning by inference. However, a certain amount of direct confirmation of such changes is available.

Among the common poisons of enzyme systems, heavy metals are accountable for ST segment depression. Definite changes in the ST segment have been encountered following intravenous injection of mercurial diuretics[39,49] a few hours after injection of neoarsphenamine,[84] and following treatment with antimony.[85-87] Heavy metals act like sodium, inhibiting enzyme systems in the Krebs cycle (aconitase, isocitric enzyme) and in fatty acid degradation.[88]

The experimental injection of mono-iodo-acetic acid, which inhibits glucose metabolism by blocking trioxophosphodehydrase, was found to induce ST segment depression.[89]

In the above examples, enzyme system blockage limits aerobic metabolism and ST segment depression may result. When the suppression of enzymatic reactions is more pronounced, ST segment elevation may occur. Cyanides, for example, suppress all oxidative tissue presses and when given in large doses, ST segment elevation can be expected.[90] The same result has been observed in some cases of veratrine intoxication.[12] On the other hand, in patients with angina pectoris, the injection of cytochrome C,[91] which acts as a catalyst for oxidation, prevents the ST segment changes customarily found when these patients are subjected to exercise or low oxygen pressures.

The problem of oxidation also enters into the probable explanation of ST segment deviation occurring in various vitamin deficiencies. It is

known, for example, that deficiencies of the Vitamin B group inhibit dehydrogenase and decarboxylase, thus affecting the aerobic breakdown of carbohydrates. Experimentally induced thiamine deficiency in dogs produced a depletion of myocardial glycogen stores and ST segment elevation; this was reversed after thiamine was added to the diet.[92,93] In clinical reports ST segment depression has been occasionally ascribed to avitaminosis B_1.[94]

Depression of the ST segment in some cases of pellagra was corrected by treatment with niacin. In some cases of myocarditis, angina pectoris, and atypical hypothyroidism, the depressed ST segment was corrected by administration of niacin.[95] Also experimentally, a diet free of pantothenic acid has produced ST segment depression.

Discussion

In this report, mainly changes in extracellular concentrations, or transmembrane gradients, of potassium and sodium ions have been taken into consideration. It is suggested that in many different clinical conditions variations in serum cationic concentration play an important part in producing ST segment depression or elevation. However, other ions, or as yet unknown factors may also be involved. There are also some exceptions which do not fit the concept of sodium and potassium electrolyte gradients presented in this paper.

Shifts of potassium and sodium ions produced ST segment changes in cases of myocardial ischemia identical to those found in the experimental and clinical conditions cited in this paper. Potassium shifting into the cell, while sodium moves into the extracellular compartment, results in ST segment depression, as observed in the classic form of angina; potassium leaving the cell, while sodium enters, leads to ST segment elevation, as observed in the variant form of angina or early myocardial infarction.

Electrocardiographic changes in ischemia can probably be explained on the basis of specific alterations in the metabolism of the ischemic cell, which in turn affect intracellular and extracellular electrolyte distribution.

SUMMARY

ST segment deviations (elevation and depression) can be indicative of myocardial ischemia or injury secondary to coronary artery disease. In a wide variety of clinical conditions, however, these ST changes are "non-ischemic," reversible by appropriate therapy and should not be interpreted as pathognomonic of coronary artery disease. Clinical conditions showing these ST changes without myocardial ischemia are reviewed in this paper.

It is postulated that ST segment deviation, whether or not the result of myocardial ischemia is related to changes in potassium and sodium gradients across the myocardial cell membrane. It is further postulated that an increased transmembrane gradient of either of these ions produces ST segment depression and that a decreased transmembrane gradient produces ST segment elevation. Clinical and experimental evidence is presented supporting these postulates. However, there is little doubt that factors other than sodium and potassium are involved.

RESUMEN

Las desviaciones del segmento ST (elevación y depresión) pueden ser indicadoras de isquemia del miocardio o lesión secundaria a la enfermedad coronaria.

En una gran variedad de estados clínicos sin embargo estos cambios de ST son "no isquémicos," reversibles por la terapia adecuada y no deben interpretarse como patognomónicos de enfermedad coronaria. Las condiciones clínicas que muestran estos cambios de ST sin isquemia del miocardio se pasan en revista en este trabajo.

Se supone que la desviación del segmento ST, sea o no resultado de la isquemia miocárdica, está en relación con los cambios del potasio y del sodio en sus gradientes a través de la membrana cellular del miocardio. Se supone además que un gradiente creciente a través de la membrana, de cualquiera de estos iones produce una depresión del segmento ST y que el decrecimiento del gradiente a través de la membrana produce una elevación del Segmento ST. Se presentan evidencias clínicas y experimentales que sustentan esta interpretación. Sin embargo, no hay duda de que existen otros factores además del sodio y del potasio.

RESUMÉ

Les déviations du segment ST (élévation et dépression) peuvent indiquer une ischémie myocardique ou une altération secondaire à une affection de l'artère coronaire. Dans une grande diversité de conditions cliniques, cependant, ces modifications

du segment ST sont "non-ischémique," réversibles par une thérapeutique appropriée, et devraient être interprétées comme caractéristiques de l'atteinte de l'artère coronaire. L'auteur passe en revue les conditions cliniques montrant ces modifications du segment ST sans ischémie myocardique.

Il admet le postulat que la déviation du segment ST, qu'elle soit ou non le résultat d'une ischémie myocardique, est en rapport avec les modifications des gradients de potassium et de sodium à travers la cellule membraneuse myocardique. Il part ensuite du fait qu'une augmentation du gradient à travers la membrane de l'un de ces ions produit une dépression du segment ST et que la diminution du gradient produit une élévation du segment ST.

L'auteur apporte une preuve clinique et expérimentale à l'appui de ces postulats. Cependant, il y a une certaine possibilité pour que d'autres facteurs que le sodium et le potassium entrent en ligne de compte.

ZUSAMMENFASSUNG

Abweichungen der ST—Strecke (Hebung und Senkung) können ein Anzeichen sein für eine Ischämie des Myocards oder eine Schädigung im Gefolge einer Kranzarterienerkrankung. In einer Vielzahl von klinischen Krankheitszuständen sind jedoch diese Veränderungen des ST—Stückes "nicht ischämisch," sind reversibel durch eine geeignete Therapie und dürfen nicht als pathognomonisch für eine Kranzarterienerkrankung angesehen werden. Klinische Krankheitszustände, die diese ST-Veränderungen ohne Ischämie des Myocards zeigen, werden in dieser Mitteilung ausgewertet.

Es wird die Forderung aufgestellt, daß eine Abweichung der ST-Strecke, sei sie nun die Folge einer Ischämie des Myocards oder nicht, in Beziehung gesetzt wird zu Veränderungen im Calium- und Natrium-Druckgefälle durch die Herzmuskel-Zell-Membran. Es wird weiter als gegeben vorausgesetzt, daß ein erhöhtes transmembranes Druckgefälle eines dieser Ionen eine Senkung des ST-Stückes bewirkt, und daß ein herabgesetztes transmembranes Druckgefälle zu einer Hebung des ST-Stückes führt. Es werden klinische und experimentelle Zeugnisse zur Unterstützung dieser Postulate angeführt. Wenig Zweifel besteht jedoch darüber, daß noch andere Faktoren außer dem Natrium und Calium im Spiele sind.

Complete reference list will appear in the reprints.

FDA RULING ON CHLORAMPHENICOL

Commissioner of Food and Drugs George P. Larrick has announced (January, 1961) that a panel of distinguished physicians appointed by the National Research Council at the FDA's request has found that the antibiotic, Chloromycetin (chloramphenicol) is a valuable drug that should remain on the market for use in treating serious infections under medical supervision both in hospitals and for treatment of patients in the home. According to the ruling of the FDA, the following is to be clearly stated:

(immediate container label)

"WARNING: Blood dyscrasias may be associated with the use of chloramphenicol. It is essential that adequate blood studies be made (see enclosed warnings and precautions)."

(enclosed insert)

"WARNING: Serious and even fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) are known to occur after the administration of chloramphenicol. Blood dyscrasias have occurred after short-term and with prolonged therapy with this drug. Bearing in mind the possibility that such reactions may occur, chloramphenicol should be used only for serious infections caused by organisms which are susceptible to its antibacterial effects. Chloramphenicol should not be used when other less potentially dangerous agents will be effective or in treatment of trivial infections such as colds, influenza, viral infections of the throat, or as a prophylactic agent."

"Precautions: It is essential that adequate blood studies be made during treatment with the drug. While blood studies may detect early peripheral blood changes, such as leukopenia or granulocytopenia, before they become irreversible, such studies cannot be relied upon to detect bone marrow depression prior to development of aplastic anemia."

The Rationale for the Treatment of Ischemic Heart Disease with Anticoagulants

Thrombosis and embolism mark the natural history of ischemic heart disease. After certain drugs were found to impede blood coagulation, they were soon used in ischemic heart disease, on the assumption that they might lessen thrombosis and embolism. The worth of anticoagulant drugs in lessening the morbidity and mortality in properly selected cases of acute cardiac infarction has been accepted increasingly by clinicians. Their place in the long-term treatment of ischemic heart disease is still hotly disputed ("Pathogenesis and Treatment of Occlusive Arterial Disease," *The Proceedings of a Conference held at the Royal College of Physicians of London*, 1959).

The effect of anticoagulant drugs in ischemic heart disease may be judged both by observations on blood coagulation and thrombosis in ischemic heart disease, and by the results of properly controlled clinical trials. There is a growing number of the latter, and I propose to outline and discuss our findings in an extensive investigation of blood coagulation in ischemic heart disease.

Investigation of Blood Coagulation in Ischemic Heart Disease

Blood coagulation was first compared in 48 patients with ischemic heart disease and in 48 normal subjects of the same age and sex (McDonald, 1957; McDonald and Edgill, 1957). All had angina pectoris on exertion; although cardiac infarction had occurred previously in some

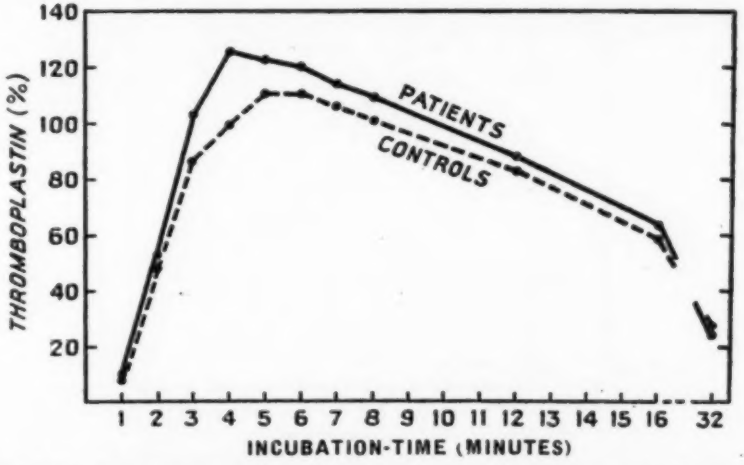

FIGURE 1: Thromboplastin-generation test: averages for patients and controls (8.5 sec. = 100 per cent). Reproduced from McDonald and Edgill (1957) by kind permission of the Editor of the *Lancet*.

of the cases, it was recent in none. The age and sex distribution were typical of ischemic heart disease. The ages were from 40 to 62 years, with average 53; there were 42 men and six women.

Tests of blood coagulation were performed on patients and controls under strictly uniform conditions, they covered the various phases of the complex series of reactions involved in blood coagulation and were the thromboplastingeneration test, platelet count and estimation of platelet-stickiness, fibrinogen estimation, two different prothrombin-times (using Russell's viper-venom, "Stypven," and brain extract, "Thrombokinase," [Geigy]), three different contact-clotting times (using ground glass, Ballotini spheres, and a silicone tube), and estimation of factor VII in plasma and serum.

When the results of these tests were compared, the mean values for patients and normals differed significantly in relation to thromboplastin-generation (Figure 1), platelet-stickiness (Figure 2), fibrinogen estimation and prothrombin-time (Russell's viper-venom, "Stypven"). These results indicated increased coagulability of the blood in the patients with ischemic heart disease, compared to the normals of the same age and sex. All these differences were significant at the 1 per cent level, except in the prothrombin-time (Russell's viper-venom, "Stypven") when the degree of significance was at the 5 per cent level. A statistically significant difference was not found between patients and controls in total platelet counts, in contact-clotting times, in estimation of factor VII in

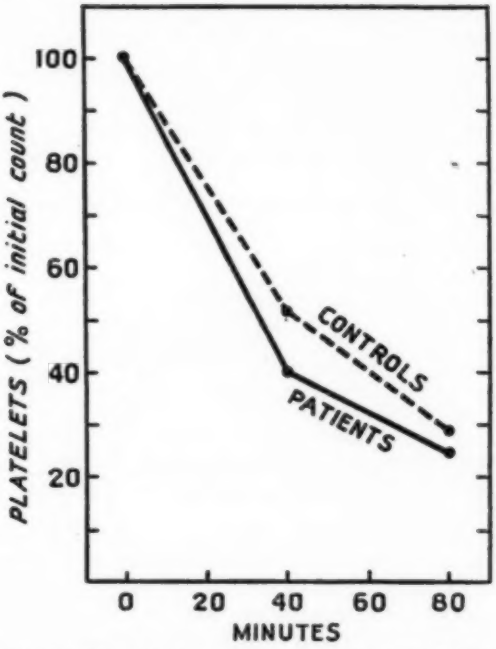

FIGURE 2: Platelet-stickiness tests: averages for patients and controls. The remaining percentage of the initial platelet count reflects the stickiness of the platelets. Reproduced from McDonald and Edgill (1957) by kind permission of the Editor of *Lancet*.

plasma and serum, nor in the prothrombin-times using brain extract ("Thrombokinase," [Geigy]). Thus for the first time, hypercoagulability of the blood in ischemic heart disease was clearly demonstrated. Subsequently these differences were found to remain unaltered on repeated testing. In a joint examination of platelet stickiness and thromboplastin-generation, in 15 paired male patients and controls, a definite relation between the two tests was established for the patients, but not for the controls (Figure 3).

Blood coagulation (with regard to thromboplastin-generation, platelet-stickiness, fibrinogen estimation, and prothrombin time using "Stypven") was next compared in normal subjects and in patients with angina pectoris on exertion, acute coronary insufficiency, and recent cardiac infarction (McDonald and Edgill, 1959). Mean values for platelet-stickiness, fibrinogen estimation and thromboplastin-generation significantly increased from normal subjects to patients with angina pectoris, and from these to patients with cardiac infarction (Fig. 4). All these differences were significant at the 1 per cent level, except the difference between the mean values for patients with angina pectoris and cardiac infarction which was nearly significant at the 5 per cent level. Findings in acute coronary insufficiency were not significantly different from those in angina pectoris, except that the platelet count was significantly higher, at the 5 per cent level, in acute coronary insufficiency (see Table 1), than in normal subjects and in patients with angina pectoris. This

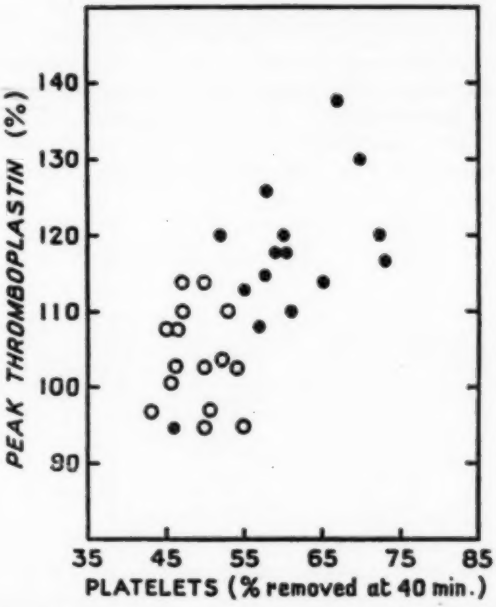

FIGURE 3: Relation between peak percentage reached in thromboplastin-generation test and platelet-stickiness (recorded here as per cent of initial platelet-count removed at 40 min. i.e. 100 minus per cent remaining): solid circles, patients; open circles, controls. Reproduced from McDonald and Edgill (1957) by kind permission of the Editor of the *Lancet*.

finding remains unexplained. The mean platelet count in patients with cardiac infarction was intermediate and did not differ significantly from the others. On repeated testing, platelet counts varied more in patients with ischemic heart disease than in normal subjects; this may be related to the increase found with acute coronary insufficiency.

These studies, therefore, confirmed the hypercoagulability of the blood that we had described previously in patients with angina pectoris, and showed that hypercoagulability was greater after cardiac infarction. The facts are in full accord with the clinical observation that patients with ischemic heart disease have an increased tendency to thrombosis.

The Significance of Hypercoagulability

Tests of blood coagulation which are performed *in vitro* must be considered in relation to coagulability and thrombosis *in vivo*. They may be assumed to reflect coagulability of blood *in vivo*, and thrombosis is likely to be associated with hypercoagulability. It would, of course, be preferable to study thrombosis within the patient, if it were possible to do so. Impaired fibrinolysis and thrombolysis may yet prove to be of paramount importance. Absolute figures in any of the tests of blood coagulation may permit only limited interpretation; the comparison of normal subjects with patients, or of patients in different phases of their disease, is more valuable. Increased platelet stickiness may reflect merely the presence of intravascular thrombosis (Bobek and Cepelak, 1958), and we have found it increased in extracardiac thrombotic conditions.

An important question is whether hypercoagulability is a cause or an effect of ischemic heart disease. Relative hypercoagulability of the blood exists in some normal people, but there is no evidence as yet that this predisposes to ischemic heart disease. Hypercoagulability may well be an effect of ischemic heart disease rather than a cause. This is not to deny that temporary hypercoagulability, followed by thrombosis, might not initiate the disease, nor that previous hypercoagulability might not increase with it.

There are at least three possible reasons for hypercoagulability after recent cardiac infarction. A phase of hypercoagulability may precede immediately infarction, initiate coronary thrombosis, and persist after

TABLE 1—PLATELET-COUNTS IN NORMAL SUBJECTS AND IN PATIENTS WITH ANGINA PECTORIS, ACUTE CORONARY INSUFFICIENCY, AND CARDIAC INFARCTION

	Number of Subjects	Mean of platelet-counts, per c.mm. whole blood, in thousands*	Standard error	Pooled standard deviation within group
Normal subjects	22	256.0	10.7	50.2
Angina pectoris	30	245.7	9.2	
Acute coronary insufficiency	7	314.4	19.0	
Cardiac infarction	10	280.5	15.9	

*For significance of differences see text.

the acute episode. On the other hand, hypercoagulability may reflect merely the presence of recent thrombosis, or be caused by tissue damage and intravascular stasis. The natural history of ischemic heart disease could be affected by hypercoagulability of the blood in different ways. Thus, an increased tendency to mural thrombosis, leading to atherosclerosis (Duguid, 1946), and a greater likelihood of complete arterial occlusion by thrombosis, would be expected if hypercoagulability is associated with an increased tendency to thrombosis.

Action of Fats

It has been thought likely that the activity of platelets in blood coagulation is due to ethanolamine phosphatide or some related compound (O'Brien, 1956), but for the present, it may be accepted that "not enough is known about the connection between clotting and thrombosis to decide whether or not the part played by fats in blood coagulability is relevant to the mechanism of thrombosis" (Poole, 1958). Modification of the diet may, however, produce an anticoagulant effect. Diminished platelet stickiness and lowered serum-cholesterol have been found after the rice-fruit diet (McDonald and Edgill, 1958), but no evidence was found that the change in stickiness was due to the reduction of cholesterol, as opposed to some other metabolic change. When serum-cholesterol was lowered by corn oil, in hypercholesterolemic patients, coagulability of the blood remained unaltered (McDonald, Edgill and Murdoch, unpublished).

Anticoagulants

Various anticoagulants act differently in restoring hypercoagulability of the blood towards normal. Thus, heparin rapidly depresses thromboplastin-generation to normal levels and below, and platelet-stickiness is reduced towards normal, although the effect is less. Phenindione affects

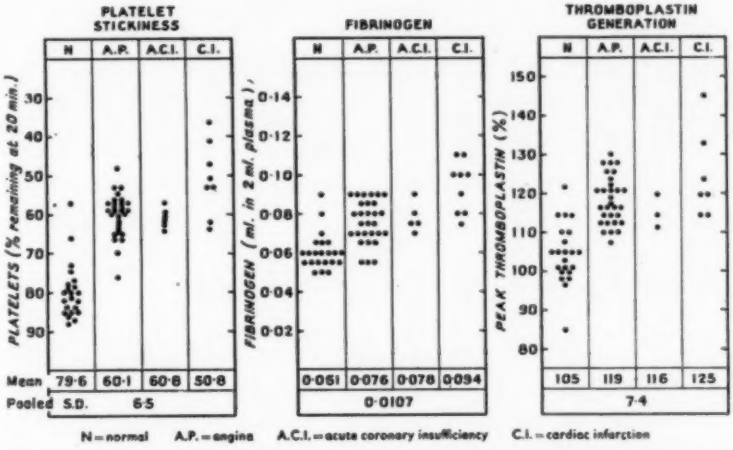

FIGURE 4: Blood coagulability with regard to platelet-stickiness, fibrinogen estimation, and thromboplastin-generation, in normals, and in patients with angina pectoris, acute coronary insufficiency and cardiac infarction. Reproduced from McDonald and Edgill (1959) by kind permission of the Editor of the *Lancet*.

both these tests less. The possibility of heparin deficiency or over-utilization in the disease is being further investigated. In anticoagulant therapy with phenindione, the optimum amount of the drug is usually judged by the prothrombin time. However, the amount of anticoagulant needed probably varies with the degree of hypercoagulability. It is not surprising that some patients, in whom anticoagulant therapy with phenindione appears well controlled by the prothrombin time, develop thrombosis. Ideal anticoagulant therapy might be based on the titration of the right anticoagulant against the clotting factors which are known to be normal.

CONCLUSION

There is clear evidence of phasic hypercoagulability when the blood of patients with ischemic heart disease is studied *in vitro*. This is entirely in keeping with a variable hyperthrombotic state, and ischemic heart disease is marked clinically by episodes of thrombosis. With regard to anticoagulant therapy in the management of ischemic heart disease, it seems reasonable to assume that any measure which safely corrects hypercoagulability and returns blood coagulation to normal, should improve the prognosis.

ACKNOWLEDGEMENT: Much of the material included in this article was given in a communication to the Royal Society of Medicine, on October 29, 1959. The author gratefully acknowledges the permission of the Editor of the *Proceedings of the Royal Society of Medicine*, to reproduce it.

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London, England

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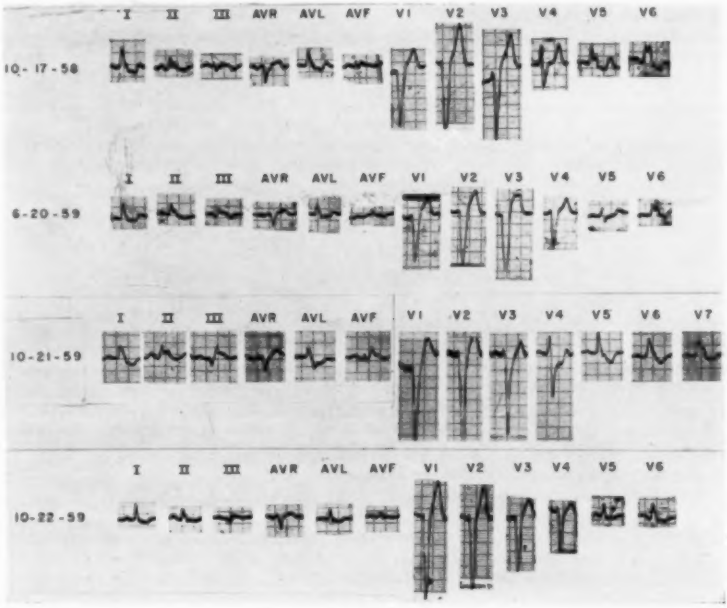
ELECTROCARDIOGRAM OF THE MONTH

Recent and Old Posterior Wall Myocardial Infarction

A 55-year-old white male was admitted to the hospital on October 21, 1959 for severe acute myocardial infarction and died ten days later in shock. He had had a posterior myocardial infarct in 1954 (see illustration dated October 17, 1958) and angina pectoris since 1957. Mild diabetes mellitus had existed for 15 years. His blood pressure had always been normal.

The electrocardiograms of October 17, 1958 and June 20, 1959 (see illustration) showed left bundle branch block (LBBB) and the old posterior infarct best seen in Leads III and aVF. The differences in QRS contour in Leads V_4 and V_5 are ascribed to changes in electrode placement. The tracing of October 21, 1959, taken on the day of hospital admission, depicts deeper Q waves in Leads III and aVF and, more importantly, significant ST segment elevation in those leads indicative of acute myocardial injury of the posterior wall. The ST segment elevation in these leads is powerful enough to reduce the T wave inversions in Leads III and aVF due to the old posterior infarct (see illustration of October 21, 1959) and to reduce the amplitude of the upright T wave in Lead V_4. As the acute myocardial injury subsides (see illustration of October 22, 1959), the T wave inversions in Leads III and aVF resume their control amplitude and the upright T wave in Lead V_4 approaches that present on June 20, 1959.



Because of the foregoing considerations, it was believed that a new posterior wall myocardial infarct had occurred close to the old posterior infarct. This type of fresh damage superimposed upon old disease, especially on the posterior left ventricular wall, often precipitates myocardial shock as happened in this patient.

Necropsy demonstrated a recent large posterolateral myocardial infarct close to an old posterolateral infarct as well as old fibrosis of the upper part of the posterior interventricular septum. This confirmed the electrocardiographic impression.

The ST segment elevation due to acute myocardial injury not only affects the inverted T waves, as mentioned above, but also the slurring on the descending limb of the QRS complex, especially well seen in Lead I. This slurring, caused by the LBBB, seems to disappear on June 20, 1959 and October 21, 1959 (see illustration), as compared with October 17, 1958. The slurring reappears again on October 22, 1959 (see illustration) where, in Lead I, the slurring of the descending limb of the QRS complex occurs just before the ST segment and must be included in measurements of the width of the QRS complex. When this is not done, the presence of LBBB may be missed.

This patient illustrates two points: (1) the diagnosis, in the presence of LBBB, of recent and old posterior myocardial infarction made antemortem; (2) how the ST segment elevations due to acute myocardial injury will alter pre-existing T wave inversions and QRS slurrings due to LBBB.

OTTO NEURATH, M.D.,* and STEPHEN R. ELEK, M.D., F.C.C.P.**
Los Angeles, California

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**Associate Clinical Professor of Medicine, University of Southern California School of Medicine, Los Angeles.

The Committee on Electrocardiography and Vectorcardiography welcomes comments. We would also be pleased to receive EKG's of exceptional interest with brief history. Please submit material to: Stephen R. Elek, M.D., chairman, 6423 Wilshire Boulevard, Los Angeles 48, California.

ATRIAL ELECTROCARDIOGRAM AS A GUIDE TO PROGNOSIS AFTER MITRAL VALVOTOMY

The atrial electrocardiogram in mitral stenosis has been examined with special reference to its value in predicting continued favorable progress after mitral valvotomy.

The tracing in 50 patients with mitral stenosis was also examined before valvotomy. The progress of 37 who remained in sinus rhythm post-operatively was followed for 4 to 8 years after operation.

The significant finding was the relationship between return to normal of the left atrial P wave after valvotomy and satisfactory clinical progress lasting 4 to 8 years.

It is concluded that persistence of an abnormal left atrial electrocardiogram after mitral valvotomy is a sensitive guide to the inadequate functional result of the operation and foretells the future deterioration likely to follow even initial clinical improvement, where only a partial valvotomy has been achieved.

Mounsey, P., "Atrial Electrocardiogram as a Guide to Prognosis after Mitral Valvotomy," *Brit. Heart J.*, XXII, 617, 1960.

CHAPTER NEWS

Alabama Chapter

The annual meeting of the Alabama Chapter will be held at the Stafford Hotel, Tuscaloosa, April 26, at which time the following program will be presented:

1:30 p.m. Registration

2:30 p.m. "Functional Paroxysmal Ventricular Tachycardia"*
Crawford W. Adams, Nashville, Tennessee
Discussor: Robert H. Yoe, Birmingham

"Differences in the Behavior of Bronchogenic Carcinoma in the Old and the Young"
Howard A. Buechner, New Orleans
Discussor: Charles J. Donald, Birmingham

"Pulmonary Function Studies Utilizing Radioactive Xenon"
Hurst B. Hatch, Jr., New Orleans
Discussor: Ben V. Branscomb, Birmingham

"Pulmonary Mycobacteriosis, with Particular Reference to Photochromogen Disease"*
Charles A. LeMaistre, Dallas
Discussor: Thomas S. Hosty, Ph.D., Montgomery

4:30 p.m. Business meeting and election of officers

5:00 p.m. Social hour

*Supported by a grant from the Merck Sharp & Dohme Postgraduate Program.

Texas Chapter

The Texas Chapter's annual meeting will be held at the Moody Convention Center, Galveston, April 23. Following is the program:

9:00 a.m. John W. Middleton, Galveston, presiding

"Fungus Studies in Tissue with Emphasis on the Use of 'Black Light' "
Lloyd R. Hershberger, San Angelo

"Prognosis of Bronchogenic Carcinoma in Relation to the Duration of Symptoms"
Donald L. Paulson, Dallas

"Roentgen Findings in Lupus Erythematosus"
Edward Troutman, Galveston

"Diseases Due to Anonymous Bacteria"
E. S. Meador, Dallas

Ninth Charles M. Hendricks Memorial Lecture
"Physiologic Techniques for Diagnosis of Esophageal Disease"
Arthur M. Olsen, Rochester, Minnesota

12:00 noon Luncheon and business meeting (Buccaneer Club)
H. M. Anderson, San Angelo, presiding
Guest Speaker: David P. Boyd, Boston, Massachusetts—"John McCrae, Physician, Pathologist, Soldier, Poet"

2:00 p.m. John C. Wiggins, Fort Worth, presiding

"Carcinoma of the Esophagus—Combined Surgery and High Voltage Therapy"
David P. Boyd, Boston

"Primary Malignant Tumors of the Chest Wall"
C. V. Brindley, Jr., Temple

"Experiences with Mitral Regurgitation"
George Iwen and E. S. Crossett, El Paso

Missouri Chapter

A Symposium on Chest Diseases, sponsored by the Southwest Chapter of the Missouri Academy of General Practice and co-sponsored by the Missouri Chapters of the College and the American Thoracic Society, as well as the Missouri State Sanatorium, will be held at the Missouri State Sanatorium, Mt. Vernon, April 8 and 9. The registration fee is $2.50. Interested physicians may write to the sanatorium for full details.

Oklahoma Chapter

The Oklahoma Chapter of the College will present its sixth annual consecutive case conference at the Western Hills Lodge, Sequoyah State Park, Wagoner, April 8 and 9. Guest speakers will be Drs. J. Maxwell Chamberlain, New York City and William B. Tucker, Washington, D. C.

Pennsylvania Chapter

The Pennsylvania Chapter, together with the Pennsylvania Trudeau Society, will meet jointly at the Pick-Roosevelt Hotel, Pittsburgh, April 20. The following program will be presented:

9:00 a.m. Morning Session—John T. Szypulski, Harrisburg, presiding
"Recurrent Actinomycotic Pleuritis"
Abraham L. Braude, Pittsburgh
"Acid-base Disturbances in Chronic Obstructive Emphysema"
Philip A. Bromberg, Pittsburgh
"Aortic Valve Replacement"
Julian Johnson and Charles K. Kirby, Philadelphia
"Effect of Smoking on Surface Active Proteins of the Lung"
Stuart Bondurant, Bloomington, Indiana
"Bronchiolar Emphysema"
Edwin R. Fisher, Pittsburgh

12:15 p.m. Joint Luncheon
Guest speaker: Horace M. Gezon, Pittsburgh

2:00 p.m. Business meeting, Pennsylvania Chapter of the College

2:15 p.m. Afternoon Session—Archibald C. Cohen, Butler, presiding
"Clinical Aspects of Pneumoconioses"
Mario Battigelli, Pittsburgh
"Radiologic Aspects of Pneumoconioses"
Eugene Pendergrass, Philadelphia
"Physical Characteristics of Dusts in Etiologies of Pneumoconioses"
Theodore Hatch, Pittsburgh
"Pathology of Pneumoconioses"
Paul Gross, Pittsburgh
"Pulmonary Physiologic Disorders in the Pneumoconioses"
Philip Hugh-Jones, London, England

BOOK REVIEWS

SUBEROSE, Lopo de Carvalho Cancella, M.D., (dissertation presented to the Faculty of Medicine, University of Lisbon)

Lopo de Carvalho Cancella is the author of a new book, SUBEROSE, published in Lisbon, Portugal. The author is the discoverer of the disease, suberosis (1949), pneumoconiosis caused by cork dust. Extensive clinical and experimental findings and other pertinent data are objectively analyzed in an 18-page English summary at the end of the 279-page book. The originality of this study and 171 excellent illustrations deserve recognition and much credit.

ANDREW L. BANYAI, M.D., F.C.C.P.

THE CHEMISTRY OF HEART FAILURE, William C. Holland, M.D., and Richard L. Klein, Ph.D., Charles C Thomas, Springfield, 1960, 112 pages, $5.50.

The authors, experienced cardiac pharmacologists, delineate the physico-chemical mechanisms of normal heart function and of the failing heart. Digitalis is discussed chemically and with reference to congestive heart failure. Fibrillation is briefly discussed. The total area covered is extensive, emphasis is put on newer developments, especially biochemical. Easily readable, it is excellent as a survey of newer experimental developments, and does this efficiently for its small size. Lack of depth is compensated for by over 300 well selected references. An excellent book for those who want to painlessly sample the research frontiers of the biochemistry of heart failure.

MYRON PRINZMETAL, M.D., F.C.C.P.

CARDIAC RESUSCITATION, J. Willis Hurst, M.D., Charles C Thomas, Springfield, 1960, 141 pages, $5.50.

This symposium, held Emory University School of Medicine, chairmanship, Dr. Hurst, is an excellent monograph relative to the problem of cardiac resuscitation. Relatively little material on the prevention of cardiac arrest. Good sketch of the internists' view by Dr. Zoll. Excellent material on respiratory resuscitation and cardiac arrest as viewed by the anesthesiologists. Greatest value is that the discussion by lawyer, theologian, and the excellent section of questions and answers are new and novel. Other material previously published in several texts and articles. Over all book, excellent. Should be in all libraries for medical schools, hospitals, and teaching institutions. Entire book, fine quality publication. Writing, interesting, accurate, concise. Valuable addition to question of resuscitation. Only criticism, does not have quite enough material for individual who wants to know how to prevent, manage the after care and therapy for cardiac resuscitation. As a symposium it is excellent, concise, enjoyable piece of work.

MAX S. SADOVE, M.D., F.C.C.P.

PROGRAM

27th ANNUAL MEETING
AMERICAN COLLEGE OF CHEST PHYSICIANS
COMMODORE HOTEL, NEW YORK CITY
JUNE 22-26, 1961

POSTGRADUATE SEMINARS
Thursday, June 22

Morning Sessions— 9:00 a.m.

AM-1 CARDIOLOGY (SURGICAL)

Chairman:

George H. Humphreys II, New York City, Chairman, Department of Surgery, Columbia-Presbyterian Medical Center

The Physiologic Criteria of Operability in Cardiac Surgery

A. Gregory Jameson, New York City, Assistant Professor of Pediatrics, Columbia University

Indications and Results of Surgical Repair for Septal Defects

Edgar P. Mannix Jr., Brooklyn, Associate Professor of Surgery, State University of New York

Follow-up Results of Surgical Repair for Valvular Heart Disease

Frank Glenn, New York City, Surgeon-in-Chief, The New York Hospital

Surgical Management for Diseased Large Arteries

Elliott S. Hurwitt, New York City, Chief, Surgical Division, Montefiore Hospital

Panel Discussion: "The Surgical Management of Cardiovascular Disease"

Moderator:

Jere W. Lord, Jr., New York City, Professor of Clinical Surgery, New York University Postgraduate Medical School

Panel:

Charles P. Bailey, New York City, Chairman and Professor of Surgery, New York Medical College
Frank Glenn, George Humphreys II, Elliott S. Hurwitt, A. Gregory Jameson and Edgar P. Mannix Jr.

AM-2 PULMONARY DISEASE (MEDICAL)

Chairman:

Oscar H. Friedman, New York City, Associate Physician for Thoracic Diseases, The Mount Sinai Hospital

Indications for Pleural and Pulmonary Biopsy

Sol Katz, Washington, D. C., Chief, Medical Service, Mt. Alto Veterans Administration Hospital

Acute Cardiorespiratory Insufficiency—Its Clinical and Therapeutic Aspects

Alvan L. Barach, New York City, Attending Physician in Medicine, Presbyterian Hospital

Considerations in the Commercial Air Transportation of Patients with Respiratory Disease

Robert B. Stonehill, Lt. Col., MC, USAF, Lackland Air Force Base, Texas, Chief, Pulmonary Disease Service, Lackland Air Force Base Hospital

The Treatment of Pulmonary Infections Due to Resistant Staphylococci

Donald B. Louria, New York City, Assistant Professor of Medicine, Cornell University

Dust Diseases of the Lungs and Their Complications

Coleman B. Rabin, New York City, Consultant Physician for Chest Diseases, The Mount Sinai Hospital

A ten minute question and answer period will follow each lecture.

Postgraduate Seminars, (Continued)

Afternoon Sessions—2:00 p.m.

PM-1 CARDIOLOGY (MEDICAL)

Chairman:

John S. LaDue, New York City, Director of Cardiology, Sloan-Kettering Institute, Memorial Center

Evaluation of Drug Therapy for Angina Pectoris

Henry I. Russek, Staten Island, Consultant in Cardiovascular Disease, U. S. Public Health Service Hospital

Management of Rheumatic Carditis

Sidney Blumenthal, New York City, Professor of Clinical Pediatrics, Columbia University

The Effects of Diet and Drugs Upon the Serum Lipids

Howard Eder, Brooklyn, Associate Professor of Medicine, State University of New York

Treatment of Heart Block

Simon Dack, New York City, Associate Clinical Professor of Medicine, New York Medical College

Auscultation: A Neglected Art

J. Scott Butterworth, New York City, Associate Professor of Medicine, New York University

The Use of Diuretics

E. Hugh Luckey, New York City, Professor of Medicine, Cornell Medical College

Treatment of Hypertension

John H. Moyer, Philadelphia, Professor and Chairman, Department of Internal Medicine, Hahnemann Medical College

A five minute question and answer period will follow each lecture.

PM-2 PULMONARY DISEASE (SURGICAL)

Chairman:

John L. Pool, New York City, Associate Thoracic Surgeon, Sloan-Kettering Institute, Memorial Center

Traumatic Thoracic Emergencies

Joseph M. Ford, New York City, Associate Attending, Chest Service, Bellevue Hospital

Congenital Deformities of the Sternum and Costal Cartilages

Charles W. Lester, New York City, Consulting Thoracic Surgeon, Hospital for Special Surgery

Difficulties in Differential Diagnosis of Mediastinal Tumors

Daniel A. Mulvihill, New York City, Chief, Section of Thoracic Surgery, St. Vincent's Hospital

The Significance of a Solitary Lung Shadow with a Primary Cancer Elsewhere

William G. Cahan, New York City, Associate Attending Surgeon (Thoracic Service), Memorial Hospital

The Prevention and Management of Pleural Complications Following Pulmonary Surgery

Lawrence Miscall, New York City, Assistant Professor of Clinical Surgery, Cornell University

A ten minute question and answer period will follow each lecture.

NOTE: These postgraduate seminars on diseases of the chest will be presented at the Commodore Hotel on Thursday, June 22, and are open to all physicians. The registration fee for each seminar is $7.50.

Registration for the seminars must be made in advance and accompanied by the tuition fee; seating capacity is limited and reservations will be accepted in the order received. A coupon for this purpose may be found on page 356. Please indicate your preference by number.

SCIENTIFIC PROGRAM
Saturday, June 24

8:55 a.m.—Scientific Session

Chairmen:

M. Jay Flipse, Miami, President
Hollis E. Johnson, Nashville, President-Elect

Cine Symposium

9:00 a.m.—Fibrinolytic Therapy in Acute Coronary Thrombosis

The Role of Selective Coronary Cine-Arteriography

F. Mason Sones, Jr., Cleveland, Director, Cardiac Catheterization Laboratory, Cleveland Clinic

The Experimental Creation and Lysis of Coronary Thrombosis

Houck E. Bolton, Chicago, Chief, Cardiovascular Surgery, Edgewater Hospital

The Intra-Arterial Use of Fibrinolysis in Acute Coronary Thrombosis—Clinical Application

Julian Ambrus, Buffalo, Assistant Professor of Pharmacology, University of Buffalo, Roswell Park Memorial Institute

The Medical Evaluation of Fibrinolytic Therapy

Thomas J. Coogan, Chicago, Clinical Associate Professor of Medicine, University of Illinois

Panel Discussion and Questions from the Floor

Moderator:

George C. Griffith, Los Angeles, Professor of Medicine, University of Southern California

10:40 a.m.—Thoracic Surgical Emergencies in Infants

Cine-Fluorography in Diagnosis of Pulmonary and Esophageal Emergencies

Harvey White, Chicago, Assistant Professor of Radiology, Northwestern University

Surgical Management of Pulmonary and Esophageal Emergencies

Johann L. Ehrenhaft, Iowa City, Professor of Surgery and Chairman, Division of Thoracic Surgery, University of Iowa

Obstructive Congenital Respiratory Anomalies

Paul H. Holinger, Chicago, Professor of Bronchoesophagology, University of Illinois

The Pediatrician's Role in Thoracic Emergencies

Milton I. Levine, New York City, Associate Professor of Pediatrics, Cornell University

Panel Discussion and Questions from the Floor

Moderator:

Roy F. Goddard, Albuquerque, Director, Department of Pediatric Research, Lovelace Foundation

NOTE: Each of the first three panelists will have ten minutes to present a film and five minutes for discussion. The fourth panelist (internist) will have ten minutes for evaluation of the preceding presentations and the remaining twenty minutes will be used by the moderator for a general discussion between the panelists and to answer questions from the floor.

12:00 noon—Round Table Luncheon Meetings (See page 345)

2:00 p.m.—Scientific Session No. 1

Chairmen:

Howard S. Van Ordstrand, Cleveland, Chairman, Pulmonary Section, Committee on Scientific Program
Charles K. Petter, Waukegan, Illinois, Superintendent and Medical Director, Lake County Tuberculosis Sanatorium

A Comparison of the Results of Simultaneous Mantoux and BCG Scarification Testing

Bernard Pollak, Montreal, Assistant Medical Director, Royal Edward Laurentian Hospital

Further Investigation on the Usefulness of the Direct Qualitative Micro-Niacin Test for Distinguishing Human Tubercle Bacilli from Other Mycobacteria: A Comparative Study of Techniques Using Fresh and Stored Cultures of Varying Ages

Maurice S. Tarshis, Alexandria, Louisiana, Director, Tuberculosis Research, Medical Research Laboratory, Veterans Administration Hospital

Newer Antibiotic Therapy for Infections of the Lower Respiratory Tract

Joseph E. Geraci, Rochester, Assistant Professor of Medicine, Mayo Foundation and University of Minnesota Graduate School

Saturday, June 24 (Continued)

Surgical Therapy of Chronic Pulmonary Histoplasmosis With and Without Amphotericin B
Oren A. Beatty, Louisville, Hospital Director; Nathan Levene, Thoracic Surgeon; N. A. Saliba, Staff Physician; and Julio Coelho, Assistant Thoracic Surgeon, State Tuberculosis Hospital

Follow-Up of the Patients Discharged with Open-Negative Cavity Syndrome
Raymond F. Corpe, Rome, Georgia, Superintendent, and Frank A. Blalock, Staff, Battey State Hospital

Ambulatory Treatment of Pulmonary Tuberculosis
S. Clive Cohen, Boston, Chief Examining Physician, Tuberculosis Program, Boston Health Department, and Edward Blacker, Department of Health, Division of Alcoholism

Evaluation of Segmental Resection of the Right Upper Lobe
Neil C. Andrews, Columbus, Associate Professor of Thoracic Surgery; Foster Marshall II, Resident in Surgery; and A. J. Christoforidis, Assistant Professor of Radiology, Ohio State University

The Influence of Antituberculous Drugs on the Anonymous Acid-Fast Organism
Dieter Koch-Weser, Cleveland, Associate Professor of Medicine, Western Reserve University

Symposium on Medical Aspects of Air Pollution

Moderator:
Seymour M. Farber, San Francisco, Chief, Tuberculosis and Chest Service University of California, San Francisco General Hospital

Epidemiology
Thomas F. Mancuso, Columbus, Chief, Division of Industrial Hygiene, Ohio State Department of Public Health

Physiology
Charles E. Schoettlin, Canoga Park, California, Occupational Health Physician, Rocketdyne Division, North American Aviation, Inc.

Particulate Matter
G. W. H. Schepers, Wilmington, Delaware, Haskell Laboratory for Industrial Medicine and Physiology, E. I. du Pont de Nemours & Company

Chemical Agents
W. C. Hueper, Bethesda, Maryland, Chief, Environmental Cancer Section, National Cancer Institute, National Institutes of Health, Public Health Service

Panel Discussion and Questions from the Floor

2:00 p.m.—Scientific Session No. 2

Chairmen:
John F. Briggs, St. Paul, Chairman, Cardiovascular Section, Committee on Scientific Program
William A. Hudson, Detroit, Chief Surgeon, Oakland County Tuberculosis Sanatorium

Circulation Studies in Newborn Lambs with Respiratory Distress
Klara J. Prec, Chicago, Assistant Professor of Pediatrics; LeClaire Leslie, Research Fellow; and Jacqueline Lax, Research Assistant, University of Chicago

Studies on the Transitional Circulation of the Newborn Infant
Mildred Stahlman, Nashville, Assistant Professor of Pediatrics; Robert Merrill, Instructor in Pediatrics, Vanderbilt University; and L. Stanley James, Assistant Professor of Pediatrics, Columbia University

Influence of the Delivery Process on the Cardiovascular System of the Newborn
L. Stanley James, New York City, Assistant Professor of Pediatrics, Columbia University

Cardiac Surgery in the Newborn
Peter V. Moulder, Chicago, Associate Professor of Surgery, University of Chicago

Present Status of Therapy in Rheumatic Myocarditis
Alvan R. Feinstein, Irvington-on-Hudson, New York, Medical Director, Irvington House

Virus Myocarditis in Infancy and Childhood
Henry R. Shinefield, New York City, Assistant Professor of Pediatrics, Cornell University

The Pathology of Myocarditis
William C. Manion, Washington, D. C., Chief, Cardiovascular Pathology, Armed Forces Institute of Pathology

Experience of Open Heart Surgery in a Community Hospital
Clair E. Basinger, Grand Rapids, Michigan, and Richard A. Rasmussen, Department of Thoracic Surgery, Blodgett Memorial Hospital

Panel Discussion and Questions from the Floor
Moderator:
Donald E. Cassels, Chicago, Professor of Pediatrics, University of Chicago

Sunday, June 25

9:00 a.m.—Scientific Session No. 1

Chairmen:

Oliver K. Niess, Major General, USAF, Washington, D. C., The Surgeon General, U. S. Air Force

Alfred Goldman, St. Louis, Associate Professor of Clinical Medicine, Washington University

The New and the Old in the Treatment of Hypertension

Moderator:

Grace M. Roth, Albuquerque, Consultant, Vascular Laboratory, Lovelace Clinic

The Effects of Hydralazine, Reserpine and Veratrum on Hypertension

Edward D. Freis, Washington, D. C., Senior Medical Investigator, Veterans Administration Hospital

Chlorothiazide (Thiazide Diuretics)

Walter M. Kirkendall, Iowa City, Professor of Medicine, State University of Iowa

Guanethidine

John H. Moyer, Philadelphia, Professor and Chairman, Department of Internal Medicine; and Albert N. Brest, Director, Hypertensive-Renal Unit, Department of Internal Medicine, Hahnemann Medical College

Renovascular Hypertension

Morton H. Maxwell, Los Angeles, Associate Clinical Professor of Medicine, University of California

Panel Discussion and Questions from the Floor

What's New in Cardiovascular Surgery

Moderator:

Charles P. Bailey, New York City, Professor and Chairman, Department of Surgery, New York Medical College

Surgical Treatment of Aortic Regurgitation

Earle B. Kay, Cleveland, Chief, Thoracic and Cardiovascular Surgery; H. A. Zimmerman, Chief of Cardiology; Akio Suzuki, Research Fellow; and David Mendelsohn, Chief, Department of Anesthesia, St. Vincent's Charity Hospital

Open Heart Surgery at Profound Levels of Hypothermia

Gumersindo Blanco-Dalmau, Philadelphia, Instructor in Thoracic Surgery and Director, Cardiac Surgery Research, Hahnemann Medical College and Hospital

Thromboendarterectomy for Coronary Heart Disease

Charles P. Bailey, New York City, and William Lemmon, New York Medical College

Treatment of Transposition of the Great Vessels by Transposing the Venous Return

Thomas G. Baffes, Chicago, Associate in Surgery, Northwestern University

Panel Discussion and Questions from the Floor

9:00 a.m.—Scientific Session No. 2

Chairmen:

Burgess L. Gordon, Chicago, Visiting Physician, Jefferson Medical College (Philadelphia)

Joseph F. Tomashefski, Columbus, Assistant Professor of Medicine and Physiology, Ohio State University

Pulmonary Interstitial Emphysema and Its Sequelae in the Newborn Infant

Paul A. Kirschner, New York City, Assistant Attending Surgeon, and Lotte Strauss, Division of Pediatric Pathology, Mount Sinai Hospital

Pre-Emphysema in Children—Its Recognition and Treatment

Roy F. Goddard, Albuquerque, Director, Department of Pediatric Research, Lovelace Foundation

Congenital Unilobar Emphysema: A Clinical and Pathologic Study of Surgically Treated Cases

Robert D. Mercer, Cleveland, Department of Pediatrics; William Hawk, Department of Pathology; and Ghazar Darakjian, Department of Pediatrics, Cleveland Clinic

The Pathology of Industrial Emphysema

G. W. H. Schepers, Wilmington, Delaware, Haskell Laboratory for Industrial Medicine and Physiology, E. I. du Pont de Nemours & Company

Patterns of Gas Exchange During Exercise in Patients with Diffuse Obstructive Pulmonary Emphysema

John W. Vance, Buffalo, Director, Emphysema Research Clinic, Chronic Disease Research Institute, University of Buffalo

Bronchography and Bronchoscopy in Emphysema and Chronic Bronchitis: Demonstration of Morphologic Changes and an Attempt at Appraisal of Severity of Functional Impairment

John H. Hirschfeld, Baltimore, Endoscopist, Associate Physician in Medicine; Otto C. Brantigan, Chief Surgeon; Milton B. Kress, Attending Physician in Medicine, in charge of Pulmonary Laboratory; Rolando V. Goco, Fellow, Cardio-Pulmonary Research, The Church Home and Hospital

Sunday, June 25 (Continued)

Intermittent Positive Pressure Breathing, Tracheostomy, and the Emphysematous Comatose Patient

Harold A. Lyons, Brooklyn, Professor of Medicine; M. Pia; Gloria Torres; and William H. Becker, State University of New York, Downstate Medical Center

Factors Influencing the Effectiveness of Intermittent Positive Pressure Breathing Therapy

Theodore H. Noehren, Buffalo, Assistant Professor of Medicine, and Paul T. Schnatz, Department of Medicine, University of Buffalo

Idiopathic Fibrous Mediastinitis

Lorenzo Hache, Rochester, Fellow in Surgery; Philip E. Bernatz, Consultant, Section of Surgery; and Lewis B. Woolner, Consultant, Section of Surgical Pathology, Mayo Clinic

The Pleuro-Pulmonary Manifestations of Acute Pancreatitis

Ronald Fishbein, Baltimore, Department of Surgery, Baltimore City Hospitals; Gerald P. Murphy, Department of Surgery, The Johns Hopkins Hospital; and Robert J. Wilder, Assistant Chief of Surgery, Baltimore City Hospitals

Clinical Trial of a New Respiratory Center Stimulant

Sami I. Said, Richmond, Assistant Professor of Medicine, and C. M. Banerjee, Fellow in Cardio-Pulmonary Physiology, Medical College of Virginia

Carcinoma of the Lung and Cigarette Smoking

Jack Reiss, Miami, Associate Clinical Professor of Medicine, University of Miami

Silent Gastroesophageal Reflux: An Important But Little-Known Cause of Pulmonary Complications

John Hines Kennedy, San Diego, Thoracic Surgical Service, San Diego County General Hospital

Lung Cysts: Evaluation of Patients for Surgery; Pre- and Post-Operative Pulmonary Function Studies

June M. Fisher, Iowa City, Assistant Professor of Internal Medicine, and George N. Bedell, Associate Professor of Internal Medicine, State University of Iowa

12:00 noon—Round Table Luncheon Meetings (See page 346)

2:00 p.m.—Scientific Session No. 1

Chairmen:

Arthur M. Olsen, Rochester, Minnesota, Professor of Medicine, Mayo Foundation, University of Minnesota

William E. Adams, Chicago, Professor of Surgery and Head of the Department, University of Chicago

Pulmonary Muscular Hyperplasia: A Correlation of Clinical, Pathological and Physiological Data

William Fraimow, Philadelphia, and Richard T. Cathcart, Barton Memorial Hospital and Jefferson Medical College Hospital

Costosternal Chondrodysis (A Variant of Tietze's Syndrome?)

Robert J. Carabasi, Temple, Texas; John J. Christian, Department of Medical Diseases of the Chest; and Hanes H. Brindley, Department of Orthopedics, Scott and White Clinic

Carcinoma of the Esophagus: Swallowing Restored with Minimal Surgery

Henry J. Heimlich, New Rochelle, New York, Assistant Clinical Professor of Surgery, New York Medical College

The Role of Host Resistance in the Natural History of Cancer

Russell H. Wilson, Dallas, Director of Research; James W. Finney, Chief of Microbiology Section; Alfred C. Schram, Chief of the Lipid Chemistry Section; Ernest H. Byers, Chief of the Protein Chemistry Section, Veterans Hospital; and John T. Mallams, Director, Radiation Therapy, Charles A. Sammons Department of Radiation Therapy and Nuclear Medicine, Baylor University Medical Center

Operative Pulmonary Artery Pressure Measurements as a Guide to Postoperative Management and Prognosis Following Pneumonectomy

James J. Rams, Chicago; Robert W. Harrison; Willard A. Fry; Peter V. Moulder; and William E. Adams, Department of Surgery, University of Chicago

Surgical Treatment of Pectus Excavatum Utilizing an Adhesive Hemicast

Colin S. Dafoe, Edmonton, Lecturer, Thoracic Surgery, and Colin A. Ross, Sessional Instructor in Thoracic Surgery, University of Alberta

Newer Applications of Pulmonary Angiography

Osler A. Abbott, Atlanta, Associate Professor of Surgery, and Britt B. Gay, Department of Thoracic Surgery, Emory University

A New Procedure for Treatment of Bronchopleural Fistula

Victor Manuel Betancourt, Mexico City, Professor of Clinical Medicine and Javier Garcia Zepeda (Histopathology), National University of Mexico

Sunday, June 25 (Continued)

A Re-Evaluation of Bronchiectasis Using Fume Fixation

William Hentel, Albuquerque, Chief, Laboratory Service; **A. N. Longfield,** Chief, Pulmonary Disease Service; and Joseph Gordon, Consultant, Thoracic Surgery, Veterans Hospital

Pulmonary Aspects of Some Toxic Experimental Space Fuels and Related Agent

Edward M. Cordasco, Niagara Falls, New York, Director, Pulmonary Function Laboratory, Mt. St. Mary's Hospital; Roger Cooper, Houston, Fellow in Medicine, Baylor University; James Murphy, Niagara Falls, Chief, Department of General Practice; and Gerald Cacio, Niagara Falls, Mt. St. Mary's Hospital

2:00 p.m.—Scientific Session No. 2

Chairmen:

Thomas W. Mattingly, Washington, D. C., Director of Medical Education, Washington Hospital Center

Donald R. McKay, Buffalo, Associate Clinical Professor of Medicine, University of Buffalo

The Importance of Atrio-Ventricular Dissociation in the Diagnosis of Digitalis Intoxication

Alfred Soffer, Rochester, New York, Chief, Cardiopulmonary Laboratories, The Rochester General Hospitals, Westside and Northside Divisions

Digitalis in Experimental, Surgically Induced Heart Block

Vallee L. Willman, St. Louis, Assistant Professor of Surgery; Theodore Cooper, Assistant Professor of Surgery; H. S. Howard, Fellow in Cardiovascular Surgery; and C. Rollins Hanlon, Professor of Surgery, St. Louis University

Hemodynamic Studies Before and After Iatrogenic Hypothyroidism in the Treatment of Severe Heart Failure

Joseph M. Merrill, Nashville, Veterans Hospital, and John H. K. Vogel, Denver, University of Colorado

The Effects of Reduced Cardiac Sympathetic Tone on Myocardial Function in Dogs

John W. Eckstein, Iowa City, Associate Professor of Internal Medicine, and A. W. Horsley, Research Fellow in Internal Medicine, State University of Iowa

The Value of the Esophageal Motility Test in the Evaluation of Chest Pain Problems

C. D. Schmidt, Rochester, Fellow in Medicine; H. D. Jones,; J. C. Hunt; C. F. Code; M. W. Anderson; and H. A. Andersen, Section of Medicine, Mayo Clinic

Coronary Blood Flow as Measured by the Electromagnetic Flowmeter

Robert J. Hall, Maj., MC, USA, Washington, D. C.; Edward M. Khouri; and Donald Gregg, Walter Reed Army Institute of Research and Walter Reed General Hospital

Hemodynamic Correlation with the Electrocardiographic Pattern of Left Ventricular Hypertrophy—Diastolic Overloading

Jerry J. Lasser, New York City, Research Fellow in Cardiology, and Richard P. Lasser, Assistant Attending Physician for Cardiology, The Mount Sinai Hospital

Electrocardiographic Patterns of Injury, Ischemia, and Necrosis

Cesar A. Caceres, Washington, D. C., Chief, Instrumentation Unit, Heart Disease Control Program, Division of Special Health Services, U. S. Department of Health, Education, and Welfare

Primary Myocardial Disease

Noble O. Fowler, Cincinnati, Associate Professor of Medicine; Mosche Gueron, Fellow in Cardiology; and David T. Rowlands, Jr., Resident in Pathology, University of Cincinnati

Coordinated Postsystolic Myocardial Augmentation Combined with Systolic Neutralization: Developmental and Clinical Applications to the Failing Heart

David H. Watkins, Denver, Chief of Surgical Service; E. R. Duchesne; and Byron E. Pollock, Division of Surgery, Denver General Hospital

Maintenance of Adequate Circulation by Mechanical Compression During Cardiac Standstill Using the Mechano-Cardiac Pulsator

Philip Y. Attalla, Chicago, Instructor and Research Associate; Cesar B. Vial, Research Fellow; Peter V. Moulder, Associate Professor of Surgery; and William E. Adams, Professor of Surgery and Head of the Department, University of Chicago

JOINT MEETING
of the
SECTION ON DISEASES OF THE CHEST
AMERICAN MEDICAL ASSOCIATION
and the
AMERICAN COLLEGE OF CHEST PHYSICIANS

New York Coliseum, Room A

Monday, June 26, 8:30 a.m.

Presiding:
Herman J. Moersch, Rochester, Minnesota, Chairman, Section on Diseases of the Chest, American Medical Association, and Director, Education and Research, American College of Chest Physicians

8:30 a.m.—*Business Meeting*

9:00 a.m.—*Chairman's Address*

"Significance of Research in Cardiopulmonary Disease"
Herman J. Moersch, Rochester

Symposium on New Approaches in the Treatment of Acquired Heart Disease

Moderator:
Arthur M. Master, New York City, Consultant Cardiologist, Mount Sinai Hospital

Isoenzymes in Coronary Disease
Felix Wroblewski, New York City, Assistant Professor of Clinical Medicine, Cornell University

Cholesterol Lowering Agents and Thyroid Analogues in Arteriosclerotic Heart Disease
William Hollander, Boston, Evans Memorial Hospital

The Treatment of Arrhythmias
Eliot Corday, Beverly Hills, Assistant Clinical Professor of Medicine, University of California

When Should the Patient with Acquired Valvular Heart Disease be Operated Upon?
Howard B. Burchell, Rochester, Professor of Medicine, Mayo Foundation Graduate School, University of Minnesota

Panel Discussion by Members of the Symposium

Tuberculosis in General Hospitals
Irving J. Selikoff, Paterson, Associate Attending Physician for Thoracic Diseases, Mount Sinai Hospital

Panel Discussion on Steroid Treatment in Pulmonary Disease

Moderator:
Sol Katz, Washington, D. C., Associate Professor of Medicine, Georgetown University

Tuberculosis
M. Henry Williams, Jr., New York City, Associate Professor of Medicine and Physiology, Albert Einstein College of Medicine

Asthma
Clarence S. Thomas, Nashville, Professor of Clinical Medicine, Vanderbilt University

Granulomatous and Collagen Diseases
Norman Hepper, Rochester, Mayo Clinic

Chronic Bronchitis and Emphysema
Alvan L. Barach, New York City, Attending Physician in Medicine, Presbyterian Hospital

Discussion and Questions from the Floor

12:00 noon—*Round Table Luncheon Meetings* *(See page 347)*
Park Sheraton Hotel

Monday, June 26, 2:15 p.m.

Presiding:

Hollis E. Johnson, Nashville, Professor of Clinical Medicine, Vanderbilt University

Symposium on Modern Diagnostic Measures in Cardiac Disease

Moderator:

George C. Griffith, Los Angeles, Professor of Medicine, University of Southern California

Renaissance of Auscultation and Phonocardiography as Diagnostic Tools
W. Proctor Harvey, Washington, D. C., Associate Professor of Medicine, Georgetown University

Advances in Electrocardiography and Vectorcardiography for the Clinical Diagnosis of Heart Disease
George E. Burch, New Orleans, Chairman, Department of Medicine, Tulane University

When Should Fluoroscopy, Angiocardiography and Cardiac Catheterization be Employed
Daniel Lukas, New York City, Associate Professor of Medicine, Cornell University

New Criteria for the Clinical Application of the Two-Step Test. Obviation of the False Negative and the False Positive
Arthur M. Master, New York City, Consultant Cardiologist, and Isadore Rosenfeld, Mount Sinai Hospital

Panel Discussion by Members of the Symposium

Scientific Papers on Pulmonary Disease

Resection of Carotid Body (Cervical Glomectomy) for Asthma
Richard H. Overholt, Boston, Surgeon, Overholt Thoracic Clinic

The Emotional Pattern Causing Cardiac and Pulmonary Disability in Professional Men and Executives
John F. Briggs, St. Paul, Associate Professor of Clinical Medicine, University of Minnesota

The Results of Operative and High Voltage Radiation Treatment in a Series of 628 Cases of Carcinoma of the Lung
David P. Boyd, Boston, Division of Thoracic Surgery, Lahey Clinic

Allergic Shock and Its Prevention
George L. Waldbott, Detroit, Senior Physician, Harper Hospital

Physiological Effects of Walking Exercise Using Oxygen in Patients with Pulmonary Emphysema
Gustav J. Beck, New York City, Instructor in Medicine; Hylan A. Bickerman, Associate Clinical Professor of Medicine, Columbia University; and Krishna Nanda, New Delhi, India

8:15 p.m.—*Fireside Conferences*
Commodore Hotel (see page 337 for Program)

SECTION ON DISEASES OF THE CHEST
JOINT MEETING WITH THE SECTION ON GENERAL PRACTICE
New York Coliseum, Room A — Tuesday, June 27, 2:00 p.m.

Presiding:

Charles R. Alvey, Muncie, Indiana, Chairman, Section on General Practice

Symposium and Panel Discussion on Angina of Effort

Moderator:

Walter Modell, New York City, Associate Professor of Pharmacology, Cornell University

This portion of the program has been arranged to provide for a critical discussion and analysis of the various aspects of the problem of angina of effort, as well as an honest evaluation of the various methods of treatment and therapeutic agents which have been recommended. Suggestions for the management of angina utilizing those procedures and agents which appear to be of greatest value will be discussed.

Nutritional and Metabolic Aspects
Frederick J. Stare, Boston, Professor of Nutrition, Harvard University

The Nitrates
J. E. F. Riseman, Boston, Assistant Clinical Professor of Medicine, Harvard Medical School

Mecamine Oxidase Inhibitors
Charles K. Friedberg, New York City, Associate Clinical Professor of Medicine, Columbia University

Sedatives and Tranquilizers
Bernard Lown, Boston, Cardiologist

Anticoagulants
Harry Beckman, Milwaukee, Professor and Chairman, Department of Pharmacology, Marquette University

Antihypertensive Agents
Arthur Grollman, Dallas, Professor of Experimental Medicine, University of Texas

Tuesday, June 27 (Continued)

Symposium on the Present Status of Newer Entities in Pulmonary Disease

Moderator:
David M. Spain, New York City, Clinical Professor of Pathology, State University of New York

Alveolar Proteinosis
Samuel H. Rosen, Washington, D. C., Senior Pathologist, Armed Forces Institute of Pathology

Pulmonary Adenomatosis, Bronchiolar and Alveolar-Cell Carcinoma
John Shapiro, Nashville, Professor of Pathology and Head of the Department, Vanderbilt, University

Hyaline Membrane Disease
Murray E. Pendleton, Boston, Instructor in Pediatrics, Harvard Medical School

Amniotic Fluid Embolization
Donald A. Nickerson, Salem, Massachusetts, Clinical Professor of Pathology, Boston University

Wegener's and Lethal Midline Granulomatosis
Edith E. Sproul, New York City, Associate Professor of Pathology, Columbia University

Cystic Fibrosis (Mucoviscidosis)
Paul A. di Sant Agnese, Bethesda, Chief, Pediatric Metabolism Branch, National Institutes of Arthritis and Metabolic Diseases, Department of Health, Education, and Welfare

Question and Answer Period by Members of the Panel

SECTION ON DISEASES OF THE CHEST
JOINT MEETING WITH THE SECTION ON RADIOLOGY

New York Coliseum, Room A

Wednesday, June 28, 9:00 a.m.

Presiding:
John P. Medelman, St. Paul, Chairman, Section on Radiology

ROENTGENOLOGY OF CARDIOVASCULAR DISEASES

Panel Discussion on Lesions of the Heart and Great Vessels

Moderator:
Conrad R. Lam, Detroit, Surgeon in Charge, Division of Thoracic Surgery, Henry Ford Hospital

Panel:
S. Gilbert Blount, Jr., Denver, Professor of Medicine, Head, Division of Cardiology, University of Colorado

F. Henry Ellis, Jr., Rochester, Associate Professor of Surgery, University of Minnesota

Melvin Figley, Seattle, Professor of Radiology, University of Washington

Richard G. Lester, Minneapolis, Assistant Professor of Radiology, University of Minnesota

Elton Watkins Jr., Boston, Assistant Clinical Professor of Surgery, Harvard Medical School

Panel Discussion on Some Fundamentals of Chest Roentgenology

Moderator:
Benjamin Felson, Cincinnati, Professor and Director, Department of Radiology, University of Cincinnati

Pathologist:
Averill Liebow, New Haven, Connecticut, Professor of Pathology, Yale University

Surgeon:
Edgar W. Davis, Washington, D. C., Clinical Professor of Thoracic Surgery, Georgetown University

Roentgenologist:
Harold G. Jacobson, New York City, Clinical Professor of Radiology, New York University

JOINT SESSION, SECTION ON DISEASES OF THE CHEST, AMA AND THE AMERICAN COLLEGE OF CHEST PHYSICIANS

Monday, June 26 — 8:15 p.m. — Commodore Hotel

FIRESIDE CONFERENCES
Subjects and Discussion Leaders

The Modern Management of Tetralogy of Fallot

Moderator:

Dwight E. Harken, Boston, Associate Clinical Professor of Surgery, Harvard Medical School

J. Maxwell Chamberlain, New York City, Associate Clinical Professor of Surgery, Columbia University

J. Francis Dammann, Jr., Charlottesville, Associate Professor of Surgical Cardiology, University of Virginia

Milton V. Davis, Dallas, Clinical Instructor in Surgery, University of Texas Southwestern Medical School

F. Henry Ellis, Jr., Rochester, Associate Professor of Surgery, University of Minnesota

Elmer C. Rigby, Los Angeles, Thoracic Surgeon, Queen of Angels Hospital

The Management of Emphysema

Moderator:

Alvan L. Barach, New York City, Attending Physician in Medicine, Presbyterian Hospital

Otto C. Brantigan, Baltimore, Chief Surgeon, Church Home and Hospital

Harold A. Lyons, Brooklyn, Professor of Medicine, State University of New York

George R. Meneely, Nashville, Associate Professor of Medicine, Vanderbilt University

John Rankin, Madison, Associate Professor of Medicine, University of Wisconsin

What's New in Cardiovascular Roentgenology

Moderator:

Benjamin Felson, Cincinnati, Professor and Director, Department of Radiology, University of Cincinnati

Andre J. Bruwer, Tucson, Radiologist, Tucson Medical Center

Thomas J. Coogan, Chicago, Clinical Associate Professor of Medicine, University of Illinois

Earl K. Shirey, Cleveland, Department of Pediatric Cardiology and the Cardiac Laboratory, Cleveland Clinic Foundation

Chemotherapy of Tuberculosis

Moderator:

Edward Dunner, Washington, D. C., Associate Director, Research Service, Veterans Administration

James O. Armstrong, Dallas, Chief of Tuberculosis Service, Parkland Hospital

Harold G. Curtis, Cleveland, Assistant Clinical Professor of Medicine, Western Reserve University

Miguel Jimenez, Mexico City, Clinical Professor of Respiratory Diseases, University of Mexico

Carl Muschenheim, New York City, Professor of Clinical Medicine, Cornell University

David Reisner, Chicago, Chief, Tuberculosis Service, Hines Veterans Administration Hospital

Fibrinolytic Agents in the Treatment of Cardiovascular Disease

Moderator:

John S. LaDue, New York City, Director of Cardiology, Sloan-Kettering Institute, Memorial Center

Houck E. Bolton, Chicago, Chief, Cardiovascular Surgery, Edgewater Hospital

William T. Foley, New York City, Associate Professor of Clinical Medicine, Cornell University

Angelo M. May, San Francisco, Adjunct Surgery, Mt. Zion Hospital

Joseph F. Uricchio, Philadelphia, Assistant Professor of Medicine, Hahnemann Medical College

The Technical Aspects of Image Amplification

Moderator:

Israel Steinberg, New York City, Associate Professor of Clinical Medicine and Radiology, Cornell University

David M. Gould, Denver, Professor of Radiology and Head of the Department, University of Colorado

John P. Medelman, St. Paul, Clinical Associate Professor of Radiology, University of Minnesota

F. Mason Sones, Jr., Cleveland, Head, Department of Pediatric Cardiology and the Cardiac Laboratory, The Cleveland Clinic Foundation

Robert E. Wise, Boston, Radiologist, Lahey Clinic

Fireside Conferences (Continued)

The Cyanotic Newborn

Moderator:

Donald E. Cassels, Chicago, Professor of Pediatrics, University of Chicago
S. Gilbert Blount, Denver, Professor of Medicine, Head, Division of Cardiology, University of Colorado
Daniel F. Downing, Philadelphia, Associate Professor of Pediatrics, Hahnemann Medical College
Robert G. Ellison, Augusta, Georgia, Professor of Surgery and Chief, Division of Thoracic Surgery, Medical College of Georgia
Roy F. Goddard, Albuquerque, Director, Department of Pediatric Research, Lovelace Foundation
Robert A. Tidwell, Seattle, Director of Cardiology, Children's Hospital

Fungus Infections

Moderator:

Michael L. Furcolow, Kansas City, Kansas, Chief, Kansas City Field Station, U. S. Public Health Service
Alvis E. Greer, Houston, Professor Emeritus of Clinical Medicine, Baylor University
E. Martinez-Rivera, Bayamon, Puerto Rico, Chief, Medical Service, A. Ruiz-Soler Hospital
Buford H. Wardrip, San Jose, California, Santa Clara County Hospital
J. Lewis Yates, Mt. Vernon, Chief of Medicine, Missouri State Sanatorium

The Present Status of IPPB

Moderator:

Theodore H. Noehren, Buffalo, Assistant Professor of Internal Medicine, University of Buffalo
David W. Cugell, Chicago, Assistant Professor of Medicine, Northwestern University
Minas Joannides, Jr., St. Petersburg, Florida, Thoracic Surgeon, Mound Park Hospital
Ross C. Kory, Milwaukee, Professor of Clinical Research, Marquette University
Ernest Mills, Burlington, Director of Pulmonary Therapy, University of Vermont Hospitals
Roger H. L. Wilson, San Francisco, Assistant Head of Continuing Education in Medicine, University of California

Treatment of the Chemotherapy Failure Case

Moderator:

Karl H. Pfuetze, Chicago, Medical Director and Superintendent, Chicago State Tuberculosis Sanitarium
Max Fleishman, Omaha, Attending Physician in Tuberculosis, Veterans Administration Hospital
Dieter Koch-Weser, Cleveland, Associate Professor of Medicine, Western Reserve University
Samuel Phillips, Memphis, Chief, Pulmonary Diseases, Veterans Administration Medical Teaching Group
William A. Werner, St. Louis, Senior Instructor in Internal Medicine, St. Louis University

Mechanical Aids in the Diagnosis of Heart Disease

Moderator:

Aldo A. Luisada, Chicago, Director, Division of Cardiology, Chicago Medical School
Ivan D. Baronofsky, San Diego, Surgeon, Scripps Memorial Hospital
Daniel A. Brody, Memphis, Professor of Medicine, University of Tennessee
H. Frederic Helmholz, Jr., Rochester, Assistant Professor of Physiology, Mayo Foundation, University of Minnesota
John H. Huston, Milwaukee, Assistant Professor of Medicine, Marquette University
Arthur M. Master, New York City, Consultant Cardiologist, Mount Sinai Hospital

Pulmonary Hypertension

Moderator:

Bernard J. Walsh, Washington, D. C., Clinical Associate Professor of Medicine, Georgetown University
Sidney Blumenthal, New York City, Professor of Clinical Pediatrics, Columbia University
Denton A. Cooley, Houston, Associate Professor of Surgery, Baylor University
Simon Dack, New York City, Associate Clinical Professor of Medicine, New York Medical College
Benjamin M. Gasul, Chicago, Professor of Pediatrics, University of Illinois
Robert H. Goetz, New York City, Director of Surgical Research and Associate Professor of Surgery, Albert Einstein College of Medicine

Monday, June 26

Collagen Diseases of the Lung

Moderator:

Herman J. Moersch, Rochester, Director, Medical Education and Research, American College of Chest Physicians

William R. Eyler, Detroit, Chairman, Department of Radiology, Henry Ford Hospital

Joseph Sieracki, Danville, Pennsylvania, Director, Department of Pathology, Geisinger Memorial Hospital and Foss Clinic

Stanford K. Sweany, Chicago, Director, Respiratory Center, Hines Veterans Administration Hospital

Respiratory Acidosis

Moderator:

Reginald H. Smart, Los Angeles, Clinical Professor of Medicine, University of Southern California

Hylan A. Bickerman, New York City, Associate Clinical Professor of Medicine, Columbia University

Burgess L. Gordon, Chicago, Visiting Physician, Jefferson Medical College (Philadelphia)

Glenn E. Horton, Memphis, Instructor in Medicine, University of Tennessee

Gordon L. Snider, Chicago, Assistant Professor of Medicine, Chicago Medical School

The Spectrum of Miliary Disorders of the Lung

Moderator:

Andrew L. Banyai, Chicago, Clinical Professor of Medicine Emeritus, Marquette University (Milwaukee)

Neil C. Andrews, Columbus, Associate Professor of Thoracic Surgery, Ohio State University

Laszlo S. Arany, Excelsior Springs, Missouri, Director, Professional Services, Veterans Administration Hospital

Philip H. Narodick, Seattle, Senior Consultant, Diseases of the Chest, King County Hospital

The Staphylococcus Problem

Moderator:

Monroe J. Romansky, Washington, D. C., Professor of Medicine, George Washington University

Benson Bloom, Albuquerque, Director, Professional Services, Veterans Administration Hospital

Eugene T. McEnery, Chicago, Clinical Professor of Pediatrics, Loyola University

Harry Shubin, Philadelphia, Medical Director, Wolffe-Broad Street Hospital and Medical Center

Allan Stranahan, Albany, New York, Associate Professor of Surgery and Head, Department of Thoracic Surgery, Albany Medical College

Robert I. Wise, Philadelphia, Magee Professor of Medicine and Head of the Department, Jefferson Medical College

Inhalation Therapy

Moderator:

Albert H. Andrews, Chicago, Clinical Associate Professor of Bronchoesophagology, Department of Otolaryngology, University of Illinois

Gustav J. Beck, New York City, Instructor in Medicine, Columbia University

Herman F. Froeb, La Jolla, California, Assistant Director, Institute of Cardiopulmonary Disease, Scripps Clinic and Research Foundation

Charles A. Gordon, Halifax, Nova Scotia, Assistant Professor of Medicine, Dalhousie University

Fernand Gregoire, Montreal, Assistant Professor of Medicine, University of Montreal

Maurice S. Segal, Boston, Director, Lung Station and Department of Inhalation Therapy, Boston City Hospital (Tufts)

Pulmonary Embolism and Thrombosis

Moderator:

Alton Ochsner, New Orleans, Professor of Surgery, Tulane University

Osler A. Abbott, Atlanta, Associate Professor of Surgery, Emory University

David P. Boyd, Boston, Thoracic Surgeon, Lahey Clinic

Ira T. Johnson, Nashville, Instructor in Clinical Medicine, Vanderbilt University

Timothy Murphy, Milwaukee, Associate Clinical Professor of Medicine, Marquette University

Henry J. Stanford, Tucson, Consultant Thoracic Surgeon, Veterans Administration Hospital

Fireside Conferences (Continued)

Bronchoesophagology

Moderator:

Paul H. Holinger, Chicago, Professor of Bronchoesophagology, University of Illinois

A. Albert Carabelli, Trenton, Chief, Department of Thoracic Medicine, St. Francis Hospital

Andre Mackay, Montreal, Director, Chest Clinic, Notre-Dame Hospital

Arthur M. Olsen, Rochester, Professor of Medicine, Mayo Foundation, University of Minnesota

James J. O'Neil, Omaha, Assistant Professor of Otolaryngology, Creighton University

George S. McReynolds, Galveston, Professor and Chief, Division of Otolaryngology, University of Texas Medical Branch

Stress as a Factor in Cardiovascular Disease

Moderator:

Francis F. Rosenbaum, Milwaukee, Associate Clinical Professor of Medicine, Marquette University

Milton W. Anderson, Rochester, Consultant in Medicine, Mayo Clinic

Herman K. Hellerstein, Cleveland, Assistant Professor of Medicine, Western Reserve University

H. Easton McMahon, New York City, Assistant Clinical Professor of Medicine (Cardiology), New York Medical College

Henry I. Russek, Staten Island, Consultant in Cardiovascular Disease, U. S. Public Health Service Hospital

Ben Sommers, St. Paul, Assistant Clinical Professor of Medicine, University of Minnesota

Traumatic Heart Disease

Moderator:

Ray W. Kissane, Columbus, Professor of Medicine in Cardiology, Ohio State University

Joseph M. Hanner, Captain, MC, USN, San Diego, Executive Officer and Cardiac Surgeon, U. S. Naval Hospital

Benjamin Manchester, Washington, D. C., Assistant Clinical Professor of Medicine, George Washington University

Nathaniel E. Reich, Brooklyn, Clinical Assistant Professor of Medicine, State University of New York

John J. Sampson, San Francisco, Clinical Professor of Medicine, University of California

Harry Vesell, New York City, Attending Physician, Beth Israel Hospital

Chest Trauma

Moderator:

Gerald L. Crenshaw, Oakland, Chief of Thoracic Surgery, Contra Costa County Hospital

Robert J. Jensik, Chicago, Senior Attending Thoracic Surgeon, Presbyterian-St. Luke's Hospital

Emil A. Naclerio, New York City, Chief, Thoracic Surgical Services, Harlem Hospital

Lawrence M. Shefts, San Antonio, Texas

David H. Waterman, Knoxville, Senior Attending Thoracic Surgeon, University of Tennessee Memorial Research Center and Hospital

Occupational Diseases of the Chest

Moderator:

Frank Princi, Cincinnati, Professor of Industrial Medicine, University of Cincinnati

J. E. Martin, Jr., Elkins, West Virginia, Chief, Internal Medicine, Golden Clinic

Eugene P. Pendergrass, Philadelphia, Professor of Radiology, University of Pennsylvania

G. W. H. Schepers, Wilmington, Delaware, Haskell Laboratory for Industrial Medicine and Physiology, E. I. du Pont de Nemours & Company

Eugene L. Walsh, Chicago, Associate Professor of Medicine, Northwestern University

Monday, June 26

Biopsy Procedures in Chest Disease

Moderator:

Karl P. Klassen, Columbus, Chief, Division of Thoracic Surgery, Ohio State University Health Center

Winthrop N. Davey, Ann Arbor, Professor of Internal Medicine, University of Michigan

Seymour M. Farber, San Francisco, Chief, Tuberculosis and Chest Service, University of California, San Francisco General Hospital

John B. Grow, Denver, Chief Consultant, Thoracic Surgery, National Jewish Hospital

Lawrence J. McCormack, Cleveland, Pathologist, Cleveland Clinic

John V. Thompson, Indianapolis, Consultant in Thoracic and Cardiovascular Surgery, Community Hospital

Pleural Effusions

Moderator:

Bruce E. Douglass, Rochester, Consultant in Medicine, Mayo Clinic

Norman Arcese, Seattle, Clinical Instructor in Medicine (Chest Disease), University of Washington

Sol Katz, Washington, D. C., Associate Professor of Medicine, Georgetown University

Solomon Netzer, Tucson, Director, Professional Services, Veterans Administration Hospital

Henry C. Sweany, Mt. Vernon, Director of Research, Missouri State Sanatorium

William L. Watson, New York City, Associate Professor of Surgery, New York University

The Etiology of Hypertension

Moderator:

Grace M. Roth, Albuquerque, Consultant, Vascular Laboratory, Lovelace Clinic

Crawford W. Adams, Nashville, Assistant Clinical Professor of Medicine, Vanderbilt University

William L. Gefter, Philadelphia, William J. Mullen Professor of Medicine, Woman's Medical College

Milton Mendlowitz, New York City, Attending Physician, Mt. Sinai Hospital

Harold W. Schnaper, Washington, D. C., Assistant Professor of Medicine, Georgetown University

What is the Tubercle Bacillus

Moderator:

Alfred G. Karlson, Rochester, Consultant, Section of Bacteriology, Mayo Clinic

Thomas W. Lester, Jr., Chicago, Chief of Staff, Suburban Cook County Tuberculosis Hospital-Sanitarium

Maurice S. Tarshis, Alexandria, Louisiana, Director, Tuberculosis Research, Medical Research Laboratory, Veterans Administration Hospital

B. H. Webster, Nashville, Visiting Physician, Internal Medicine and Chest Diseases, St. Thomas Hospital

Allergic Diseases of the Respiratory Tract

Moderator:

Harry L. Rogers, Philadelphia, Assistant Clinical Professor of Medicine (retired), Jefferson Medical College

Howard A. Andersen, Rochester, Consultant, Internal Medicine and Thoracic Disease, Mayo Clinic

Clifford H. Kalb, Milwaukee, Consultant in Surgery, St. Joseph's Hospital

Nathan E. Silbert, Lynn, Massachusetts, Chief, Department of Allergic Diseases, St. Joseph's Hospital, Lowell

Clarence S. Thomas, Nashville, Professor of Clinical Medicine, Vanderbilt University

George L. Waldbott, Detroit, Senior Physician, Harper Hospital

Fireside Conferences (Continued)

Steroid Therapy in Lung Diseases

Moderator:
David B. Radner, Chicago, Director, Chest Department, Michael Reese Hospital

Alfred Goldman, St. Louis, Associate Professor of Clinical Medicine, Washington University
Homer D. Peabody, San Diego, Rees-Stealy Medical Clinic
George R. Piness, Los Angeles, Clinical Professor of Medicine, University of Southern California
John E. Rayl, Oteen, North Carolina, Assistant Chief, Surgical Service, Veterans Administration Hospital

The Lungs in Systemic Disease

Moderator:
Eli H. Rubin, New York City, Professor of Clinical Medicine, Albert Einstein College of Medicine

Orin J. Farness, Tucson, Arizona, Internist, Tucson Medical Center
Robert L. Grissom, Omaha, Professor and Chairman, Department of Internal Medicine, University of Nebraska
Jack Reiss, Coral Gables, Florida, Clinical Assistant Professor of Medicine, University of Miami
David M. Spain, New York City, Clinical Professor of Pathology, State University of New York

The Monoamine Oxidase Inhibitors in Cardiovascular Disease

Moderator:
Samuel A. Weisman, Los Angeles, Clinical Associate Professor of Medicine, University of Southern California

Louis F. Bishop, New York City, Attending Cardiologist, Veterans Hospital
Seymour L. Cole, Los Angeles, Associate Clinical Professor of Medicine, University of Southern California
David Scherf, New York City, Professor of Clinical Medicine, New York Medical College
Henry A. Zimmerman, Cleveland, Chief of Cardiology, St. Vincent's Charity Hospital

Work Evaluation in Cardiac Disease

Moderator:
Arthur C. Kerkhof, Minneapolis, Clinical Associate Professor of Medicine, University of Minnesota

Irvin Klein, New York City, Medical Director, Workmen's Compensation Board, State of New York
Eugene M. Murphey, III, Tupelo, Mississippi, Chief of Medicine, North Mississippi Community Hospital
Myron I. Segal, Hollywood, Florida, Attending Surgeon, Hollywood Memorial Hospital
Paul Sommerfreund, Westmount, Quebec, Demonstrator, Department of Clinical Medicine, McGill University

Sarcoidosis

Moderator:
Edward H. Morgan, Seattle, The Mason Clinic

David A. Cooper, Philadelphia, Professor of Medicine, University of Pennsylvania
Harold L. Israel, Philadelphia, Clinical Professor of Medicine, Jefferson Medical College
Louis E. Siltzbach, New York City, Associate Clinical Professor of Medicine, Columbia University

Hiatus Hernia

Moderator:
Donald B. Effler, Cleveland, Chief, Department of Thoracic Surgery, Cleveland Clinic Foundation

Irving B. Brick, Washington, D. C., Associate Professor of Medicine, Georgetown University
Alfred Goldman, Los Angeles, Attending Thoracic Surgeon, Cedars of Lebanon Hospital
Henry J. Heimlich, New York City, Chief, Section of Esophageal Surgery, Flower and Fifth Avenue Hospitals
Franklin R. Smith, Seattle, Clinical Associate Professor of Surgery, University of Washington
Francis M. Woods, Boston, Associate, Overholt Thoracic Clinic

Monday, June 26

Pulmonary Cavities

Moderator:

Donald R. McKay, Buffalo, Associate Clinical Professor of Medicine, University of Buffalo

Donato G. Alarcon, Mexico City, Medical Director, Sanatorio San Angel

DeWitt C. Daughtry, Miami, Assistant Clinical Professor of Surgery (Thoracic), University of Miami

Leroy Hyde, Long Beach, California, Chief, Pulmonary Disease Service, Veterans Administration Hospital

William M. Lees, Chicago, Chief of Surgery, Municipal Tuberculosis Sanitarium

Howell S. Randolph, Phoenix, Chairman, Surgical Committee, St. Luke's Hospital

Etiologic Evaluation of Pulmonary Fibrosis

Moderator:

Joseph H. Massee, Atlanta, Associate Professor of Clinical Medicine, Emory University

Charles F. Blazsik, New York City, Medical Director, St. Anthony's Hospital for Chest Diseases

W. Leonard Howard, Northville, Michigan, Superintendent, Wm. H. Maybury Sanatorium

Isadore Pilot, Chicago, Associate Professor of Medicine, University of Illinois

Edward S. Ray, Richmond, Associate Professor of Medicine, Medical College of Virginia

MOTION PICTURE PROGRAM

Following is a tentative program of films which will be shown concurrently with the scientific program on Saturday and Sunday, June 24 and 25, at the Commodore Hotel.

Surgical Correction of Primary Tumors of the Heart

Osler A. Abbott, Atlanta, Department of Surgery, Emory University

Intracavitary Cardiac Lesions

Crawford W. Adams and Harold Collins, Nashville, Departments of Medicine and Surgery, Vanderbilt University

A Simple Procedure for the Temporary Bypass of the Pulmonary Valve

Yousif D. Al-Naaman, William M. Rogers, James E. Harrison and Ali K. Maksad, New York City, Columbia University

Coccidioidomycosis

Marco Bruschi, Bakersfield, California

Valvuloplasty for Mitral Stenosis

Dwight E. Harken, Warren J. Taylor and Samuel A. Levine, Boston, Harvard Medical School and Peter Bent Brigham Hospital

Endoscopic Pathology of the Ear, Nose and Throat, the Trachea, Bronchi and Esophagus

Paul H. Holinger, Kenneth C. Johnston, Maria Ikenberg and Joseph Brubaker, Chicago

Anomalous Pulmonary Vein to the Inferior Vena Cava: The Bronchopulmonary Triad

C. Frederick Kittle, David S. Ruhe, and J. F. Crockett, Kansas City, Kansas, University of Kansas

The Repair of Atrial Septal Defect Utilizing Cardiopulmonary Bypass

G. H. Lawrence, Seattle, Department of Surgery, The Mason Clinic

Hypoxia—Indications for Oxygen Therapy

Edwin R. Levine, Chicago, Chicago Medical School and Edgewater Hospital

Cardiopexy for Coronary Heart Disease

Maurice S. Mazel and Houck E. Bolton, Chicago, Edgewater Hospital

Bronchitis and Bronchiectasis: Differentiation for Therapy

John E. Rayl, E. D. Peasley, John T. Joyner and Lawrence Mucci, Oteen, North Carolina, Veterans Hospital

Surgical Anatomy of the Pulmonary Hylum: Part I

Franklin R. Smith and Edward A. Boyden, Seattle, Departments of Anatomy and Surgery, University of Washington

Experimental Pulmonary Embolism and Embolectomy

William S. Stoney and J. Kenneth Jacobs, Nashville, Vanderbilt University

ROUND TABLE LUNCHEONS

Friday, June 23 **Hotel Commodore**

A-1 ***Diagnosis and Treatment of Curable Hypertension***

Moderator:

Travis Winsor, Los Angeles, Associate Clinical Professor of Medicine, University of Southern California

John P. Medelman, St. Paul, Clinical Associate Professor of Radiology, University of Minnesota

John H. Moyer, Philadelphia, Professor of Medicine and Chairman, Department of Internal Medicine, Hahnemann Medical College

Grace M. Roth, Albuquerque, Consultant, Vascular Laboratory, Lovelace Clinic

Maurice A. Schnitker, Toledo, Director of Medicine and Chief of Medical Service, St. Vincent's Hospital

A-2 ***The Management of Resistant Pyogenic Infections of the Lung***

Moderator:

Chester S. Keefer, Boston, Wade Professor of Medicine, Boston University

Joseph E. Geraci, Rochester, Consultant in Medicine, Mayo Clinic

Alfred Goldman, St. Louis, Associate Professor of Clinical Medicine and Director, Medical Chest Service, Washington University

Robert L. Wise, Philadelphia, Magee Professor of Medicine and Head of the Department, Jefferson Medical College

A-3 ***The Use of Steroid Therapy in Pulmonary Tuberculosis***

Moderator:

Karl H. Pfuetze, Chicago, Medical Director and Superintendent, Chicago State Tuberculosis Sanitarium

Sumner S. Cohen, Oak Terrace, Clinical Assistant Professor of Medicine, University of Minnesota

Arvine G. Popplewell, Indianapolis, Medical Director of Hospitals, Marion County General Hospital

M. Henry Williams, Jr., New York City, Associate Professor of Medicine and Physiology, Albert Einstein College of Medicine

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PREMIERE OF FILM "MAN RETURNS TO THE SEA"

Monday, June 26, New York Coliseum

"Man Returns to the Sea" **by G. Dekle Taylor, M.D., Jacksonville, Florida. (Sound, color, 29 minutes) Millions of years ago man evolved from the sea. He is now a terrestrial being and his anatomy and physiology are not modified for an aquatic environment. For thousands of years he has contemplated returning to the sea. Centuries of imagination, endeavor, and patient experimentation were necessary, however, before his ingenuity enabled him to adapt himself again to the underwater world. He has few of the structural adaptations for an aquatic or sub-aquatic environment as is seen in the porpoise, pilot whale, hippopotamus, and other aquatic animals. This film shows man's adaptation to the water by proper methods of breathing and bathing habits. The evolution of the various swimming strokes, face mask, snorkel, and self-contained underwater breathing apparatus is shown. The effect of pressure on the ear and sinuses is demonstrated. If man will understand and accept the limitations nature has placed on him in an aquatic environment he is now able to return to the sea.**

ROUND TABLE LUNCHEONS

Saturday, June 24 **Hotel Commodore**

B-1 ***Cor Pulmonale***

Moderator:
Irving Mack, Chicago, Attending Physician, Chest Department, Michael Reese Hospital

Milton W. Anderson, Rochester, Consultant in Medicine, Mayo Clinic
Harry Goldberg, Philadelphia, Director, Department of Cardiovascular Diseases, Albert Einstein Medical Center
Thomas W. Mattingly, Washington, D. C., Director of Medical Education, Washington Hospital Center

B-2 ***Anticoagulant Therapy in Cardiovascular Disease***

Moderator:
E. Sterling Nichol, Miami, Attending Cardiologist, Miami Heart Institute

Joseph F. Borg, St. Paul, Associate Clinical Professor of Medicine, University of Minnesota
Eliot Corday, Beverly Hills, Assistant Clinical Professor of Medicine, University of California
Fay A. LeFevre, Cleveland, Department of Cardiovascular Disease, Cleveland Clinic
Gerald H. Pratt, New York City, Associate Clinical Professor of Surgery, New York University

B-3 ***Current Trends in the Treatment and Prophylaxis of Rheumatic Fever***

Moderator:
Gene Stollerman, Chicago, Associate Professor of Medicine, Northwestern University

Antoni M. Diehl, Kansas City, Associate Professor of Pediatrics, University of Kansas
Raphael N. Paul, Memphis, Assistant Professor of Pediatrics, University of Tennessee
Robert F. Ziegler, Detroit, Physician-in-Charge, Division of Pediatric Cardiology, Henry Ford Hospital

B-4 ***Medical and Surgical Management of Hiatus Hernia***

Moderator:
Herman J. Moersch, Rochester, Director, Education and Research, American College of Chest Physicians

Edgar W. Davis, Washington, D. C., Clinical Professor of Thoracic Surgery, Georgetown University
Donald B. Effler, Cleveland, Chief, Department of Thoracic Surgery, Cleveland Clinic
Paul H. Holinger, Chicago, Professor of Bronchoesophagology, University of Illinois
Bernard S. Wolf, New York City, Director, Department of Radiology, Mt. Sinai Hospital

B-5 ***Occupational Pulmonary Diseases—Their Impairment Evaluation, Compensability and Treatment***

Moderator:
Peter A. Theodos, Philadelphia, Assistant Professor of Clinical Medicine, Jefferson Medical College

John W. G. Hannon, Washington, Pennsylvania, Medical Director, McIntyre Research Foundation
Oscar A. Sander, Milwaukee, Associate Clinical Professor of Medicine, Marquette University
Arthur J. Vorwald, Detroit, Professor of Medicine and Chairman, Department of Industrial Medicine and Hygiene, Wayne State University

B-6 ***The Detection and Management of Drug Resistant Tuberculosis***

Moderator:
Raymond F. Corpe, Rome, Georgia, Superintendent, Battey State Hospital

Howard A. Buechner, New Orleans, Chief of Medical Service, Veterans Administration Hospital
Marvin S. Harris, Los Angeles, Clinical Professor of Thoracic Diseases, College of Medical Evangelists
Henry C. Sweany, Mt. Vernon, Director of Research, Missouri State Sanatorium

ROUND TABLE LUNCHEONS

Sunday, June 25 **Hotel Commodore**

C-1 ***Methods of Control and Eradication of Tuberculosis***

Moderator:
Sidney H. Dressler, Denver, Chief of Staff, National Jewish Hospital

Otto L. Bettag, Glen Ellyn, Illinois, Medical Director, DuPage County Tuberculosis Association
Katharine R. Boucot, Philadelphia, Professor of Preventive Medicine, Woman's Medical College
Charles A. Brasher, Mt. Vernon, Medical Director, Missouri State Sanatorium
J. Arthur Myers, Minneapolis, Professor Emeritus, Department of Medicine and School of Public Health, University of Minnesota

C-2 ***The Management of Intractable Heart Failure***

Moderator:
George C. Griffith, Los Angeles, Professor of Medicine (Cardiology), University of Southern California

George E. Burch, New Orleans, Chairman, Department of Medicine, Tulane University
James H. Hammond, Col., MC, USAF, Bergstrom Air Force Base, Texas, U. S. Air Force Hospital
William Likoff, Philadelphia, Clinical Professor of Medicine and Head, Section of Cardiovascular Diseases, Hahnemann Medical College
David Scherf, New York City, Professor of Clinical Medicine, New York Medical College

C-3 ***The Medical Treatment of Coronary Disease***

Moderator:
Arthur M. Master, New York City, Consultant Cardiologist, Mount Sinai Hospital

Barney M. Dlin, Philadelphia, Department of Psychiatry, Temple University
John J. Sampson, San Francisco, Clinical Professor of Medicine, University of California
Joseph Uricchio, Philadelphia, Assistant Professor of Medicine, Hahnemann Medical College
Arthur M. Vineburg, Montreal, Surgeon-in-Charge, Sub Department of Cardiac Surgery, Royal Victoria Hospital

C-4 ***The Medical and Surgical Significance of Pulmonary Hypertension***

Moderator:
Robert O. Brandenburg, Rochester, Assistant Professor of Medicine, University of Minnesota

Ivan D. Baronofsky, San Diego, Surgeon, Scripps Memorial Hospital
Howard B. Burchell, Rochester, Consultant in Medicine, Mayo Clinic
Arthur Grishman, New York City, Associate Physician in Medicine for Cardiology, Mount Sinai Hospital

C-5 ***The Treatment of the Patient with Inoperable Lung Cancer***

Moderator:
Richard H. Overholt, Boston, Surgeon, Overholt Thoracic Clinic

John Boland, New York City, Radiotherapist-in-Chief, Mount Sinai Hospital
John K. Fulton, Wichita, Internist, Wichita Clinic
Robert B. Golbey, New York City, Assistant Attending Physician, Memorial Hospital
Phineas J. Sparer, Memphis, Professor of Psychiatry and Preventive Medicine, University of Tennessee

C-6 ***Recent Developments in Inhalation Therapy***

Moderator:
Edwin R. Levine, Chicago, Director, Department of Inhalation Therapy, Edgewater Hospital

Allan Hurst, Denver, Assistant Clinical Professor of Medicine, University of Colorado
James Kieran, Berkeley, Assistant Professor of Medicine, University of California
William F. Miller, Dallas, Associate Professor of Medicine, University of Texas
Joseph F. Tomashefski, Columbus, Assistant Professor of Medicine and Physiology, Ohio State University

JOINT SESSION, SECTION ON DISEASES OF THE CHEST, AMA AND THE AMERICAN COLLEGE OF CHEST PHYSICIANS

ROUND TABLE LUNCHEONS

Monday, June 26 **Park Sheraton Hotel**

D-1 ***The Office Diagnosis of Surgical Forms of Heart Disease***

Moderator:

Ben Sommers, St. Paul, Assistant Clinical Professor of Medicine, University of Minnesota

J. Francis Dammann, Jr., Charlottesville, Associate Professor of Surgical Cardiology, University of Virginia

William F. Mazzitello, St. Paul, Assistant Clinical Professor of Medicine, University of Minnesota

Joseph W. Peabody, Jr., Washington, D. C., Clinical Assistant Professor of Thoracic Surgery, Georgetown University

D-2 ***The Management of Cardiac Emergencies***

Moderator:

Henry I. Russek, Staten Island, Consultant in Cardiovascular Disease, U. S. Public Health Service Hospital

Harry Gold, New York City, Professor of Clinical Pharmacology, Cornell University

John A. Lewis, London, Assistant Professor of Medicine, University of Western Ontario

Hugh E. Stephenson, Jr., Columbia, Professor of Surgery, University of Missouri

Arthur E. Strauss, St. Louis, Assistant Professor of Clinical Medicine, Emeritus, Washington University

D-3 ***The Diagnosis and Treatment of Cardiac Arrhythmias***

Moderator:

Samuel Bellet, Philadelphia, Professor of Clinical Cardiology, University of Pennsylvania

Simon Dack, New York City, Associate Professor of Medicine, New York Medical College

Stephen R. Elek, Los Angeles, Associate Clinical Professor of Medicine, University of Southern California

Samuel W. Hunter, St. Paul, Director of Cardiac Research, St. Joseph's Hospital

Myron Prinzmetal, Los Angeles, Institute for Medical Research, Cedars of Lebanon Hospital

D-4 ***Pulmonary Function Testing in a Doctor's Office or in a Small Hospital***

Moderator:

Reginald H. Smart, Los Angeles, Clinical Professor of Medicine, University of Southern California

Albert H. Andrews, Jr., Chicago, Clinical Associate Professor of Bronchoesophagology, Department of Otolaryngology, University of Illinois

Ross C. Kory, Wood, Wisconsin, Professor of Clinical Research, Marquette University

George R. Meneely, Nashville, Associate Professor of Medicine, Vanderbilt University

John Rankin, Madison, Associate Professor of Medicine, University of Wisconsin

D-5 ***Hospital Versus Home Treatment of Pulmonary Tuberculosis***

Moderator:

David B. Radner, Chicago, Director, Chest Department, Michael Reese Hospital

Edward T. Blomquist, Washington, D. C., Chief, Tuberculosis Program, U. S. Public Health Service

Edward H. Robitzek, Staten Island, Director of Medical Service, Sea View Hospital

Irving J. Selikoff, Paterson, Associate Attending Physician for Thoracic Diseases, Mount Sinai Hospital

D-6 ***The Management of Bronchial Asthma***

Moderator:

Howard S. Van Ordstrand, Cleveland, Head, Department of Pulmonary Diseases, Cleveland Clinic

Giles A. Koelsche, Rochester, Consultant, Section of Internal Medicine and Allergy, Mayo Clinic

George S. McReynolds, Galveston, Professor and Chief of Otolaryngology, Department of Surgery, University of Texas

Lloyd D. Mayer, Lexington, Consultant in Allergy, U. S. Public Health Service Hospital

Leon Unger, Chicago, Associate Professor of Medicine, Northwestern University

Scientific Exhibits, American Medical Association
New York Coliseum, June 25-30, 1961

EDWIN R. LEVINE, Chicago
Representative to Scientific Exhibit, Section on Diseases of the Chest

SPECIAL EXHIBIT ON PHYSIOLOGIC TESTING OF CARDIAC FUNCTION

The Special Exhibit on Physiologic Testing of Cardiac Function is presented jointly by the Section on Diseases of the Chest of the American Medical Association and the Committee on Cardiovascular Physiology of the American College of Chest Physicians, with the cooperation of the Veterans Administration.

Demonstrations will be presented each morning and afternoon by panels comprised of experts in cardiovascular physiology. This exhibit has been approved by the Council on Scientific Assembly of the AMA and will be presented for the first time at the New York Coliseum. It is a **must** for every physician interested in cardiac function.

JOHN S. LaDUE, Chairman
Committee on Cardiovascular Physiology

RUDOLPH E. FREMONT, Chairman
Committee on Scientific Exhibit

Panel Conferences and Names of Participants

Routine Methods of Evaluating Congestive Heart Failure

Moderator:
James Warren, Galveston
John S. LaDue, New York City
William Poppell Jr., New York City
John J. Sampson, San Francisco

Angiography of Valvular and Coronary Disease

Moderator:
Israel Steinberg, New York City
Melvin Figley, Seattle
Benjamin M. Gasul, Chicago
John B. Schwedel, New York City
F. Mason Sones Jr., Cleveland

Cardiac Catheterization in Acquired Heart Disease

Moderator:
John Ross Jr., New York City
Noble O. Fowler, Cincinnati
Harry Goldberg, Philadelphia
Richard Gorlin, Boston
Albert Hyman, New Orleans
Daniel S. Lukas, New York City

Cardiac Catheterization in Congenital Heart Disease

Moderator:
H. J. Swan, Rochester, Minnesota
Mary A. Engle, New York City
Doris Escher, New York City
Irving Kroop, Brooklyn
Abraham Rudolph, New York City
Philip Samet, Miami Beach

Exercise Testing

Moderator:
Arthur M. Master, New York City
John S. LaDue, New York City
George Robb, New York City
Isadore Rosenfeld, New York City
Henry I. Russek, Staten Island

Phonocardiography

Moderator:
Aldo A. Luisada, Chicago
Clarence M. Agress, Beverly Hills
Rudolph E. Fremont, New York City
John J. Kelly, San Diego
Joshua Lynnfield, Brooklyn

Ballistocardiography

Moderator:
H. De Witt Smith, Princeton, N. J.
Rudolph E. Fremont, New York City
Richard Gubner, Brooklyn
S. A. Talbot, Baltimore
Nahum Winer, New York City

Vectorcardiography

Moderator:
George E. Burch, New Orleans
J. A. Abildskov, Syracuse
Arthur Grishman, New York City
W. R. Milnor, Baltimore
Hubert Pipberger, Washington, D. C.

Cardiac Evaluation as Applied to Rehabilitation

Menard M. Gertler, New York City
Herman K. Hellerstein, Cleveland
S. J. Kottke, Minneapolis
W. G. Kubicek, Minneapolis
H. Easton McMahon, New York City

Subjects of Scientific Display and Names of Contributors

Routine Methods of Evaluating Congestive Heart Failure
William Poppell Jr., New York City

Angiography of Valvular and Coronary Disease
Donald S. Littman, Little Silver, N. J., and F. Mason Sones, Jr., Cleveland

Cardiac Catheterization in Acquired Heart Disease
Richard Gorlin, Boston, Albert Hyman, New Orleans, Abraham Schaeffer, New York City, and John B. Wild, Brooklyn

Cardiac Catheterization in Congenital Heart Disease
Archer S. Gordon, Los Angeles, Albert Hyman, New Orleans, and Irving Kroop, Brooklyn

Exercise Testing
A. Freiman, J. S. LaDue and Isadore Rosenfeld, New York City

Phonocardiography
Rudolph E. Fremont, New York City, and Aldo A. Luisada, Chicago

Ballistocardiography
William Dock, Palo Alto, and Rudolph E. Fremont, New York City

Vectorcardiography
Hubert Pipberger, Washington, D. C.

Cardiac Evaluation as Applied to Rehabilitation
Menard M. Gertler, New York City, S. J. Kottke and W. G. Kubicek, Minneapolis

This exhibit was supported in part by grants from Burroughs-Wellcome Company, Inc., and Endo Laboratories, Inc.

SPECIAL EXHIBIT ON PULMONARY FUNCTION

The Special Exhibit on Pulmonary Function is presented jointly by the Section on Diseases of the Chest of the American Medical Association and the American College of Chest Physicians, with the cooperation of other medical societies.

Demonstrations will be presented throughout each morning and afternoon to show how pulmonary function tests and physiological therapy may be conducted in the hospital and in the office. The exhibit and program of demonstrations has been developed and continued with the help of many physicians.

A "lung station" suitable for a hospital or clinic is designed to aid in diagnosis, prognosis, therapy, and the evaluation of disability in pulmonary disease in much the same manner that a "heart station" serves the needs of clinicians concerned with heart disease.

Demonstrations on Testing and Physiological Therapy

Each morning and afternoon a special session of demonstrations will be held. The schedule below shows the moderators and demonstrators. Each will give a 15 minute presentation on the subject designated. These will be followed by a 30 minute period of questions and answers and of open discussion.

JOSEPH F. TOMASHEFSKI, Chairman
Committee on Pulmonary Physiology

GEORGE R. MENEELY, Chairman
Committee on Scientific Exhibit

Monday, June 26

10:00 a.m.

Moderator:
Anesthesia and Pulmonary Function
William F. Miller, Dallas

Demonstrators:
Pathogenesis of Cor Pulmonale
G. W. H. Schepers, Wilmington

Pulmonary Function in Industry
Joseph F. Tomashefski, Columbus

Pulmonary Function and Air Travel
Roger H. L. Wilson, San Francisco

2:30 p.m.

Moderator:
Tracheostomy in Emphysema
Edgar Mayer, New York

Demonstrators:
Screening Tests for Impaired Pulmonary Function
Ben V. Branscomb, Birmingham

Hospital Testing of Pulmonary Function
A. L. Loomis Bell, New York City

Surgery in Pulmonary Emphysema
Osler A. Abbott, Atlanta

Tuesday, June 27

10:00 a.m.

Moderator:
Evaluation of Pulmonary Disability
Oscar A. Sander, Milwaukee

Demonstrators:
Pathology of Pulmonary Emphysema
Hollis G. Boren, Houston

Prognosis in Pulmonary Emphysema
Joseph M. Merrill, Nashville

Blood Changes in Pulmonary Failure
Richard T. Cathcart, Philadelphia

2:30 p.m.

Moderator:
Pulmonary Compliance
Hylan A. Bickerman, New York City

Demonstrators:
Field Testing in Industry
J. W. G. Hannon, Washington, Pa.

Pulmonary Function in Children
Roy F. Goddard, Albuquerque

Selection of Cases for Surgery
Sam E. Stephenson, Jr., Nashville

Wednesday, June 28

10:00 a.m.

Moderator:
Thyroid Suppression in Pulmonary Failure
W. F. Hamilton, Augusta, Georgia

Demonstrators:
Effect of Altitude in Impaired Pulmonary Function
George W. Bower, Detroit

Pulmonary Resistance
David W. Cugell, Chicago

Gas Chromatography of Blood Gases
Lloyd H. Ramsey, Nashville

2:30 p.m.

Moderator:
Oxygen Therapy in Pulmonary Failure
Alvan L. Barach, New York City

Demonstrators:
Intrapulmonary Gas Mixing
Frank Lovejoy, Rochester, New York

Oxygen Cost of Breathing
Richard A. Bader, New York City

Diffusion Studies
James K. Alexander, Houston

Thursday, June 29

10:00 a.m.

Moderator:

Inhalation Therapy
Edwin R. Levine, Chicago

Demonstrators:

Lung Volume in Pulmonary Emphysema
Harold A. Lyons, Brooklyn

The Army-VA Cooperative Study of Pulmonary Function
Ross C. Kory, Milwaukee

Use of Gas Chromatography for Respiratory Gas Analysis
Thomas J. Ormsby, Newark

2:30 p.m.

Moderator:

Pulmonary Roentgen Kinetic Densitometry
Albert H. Andrews, Chicago

Demonstrators:

Management of Cor Pulmonale
John L. Patterson, Jr., Richmond

Organization of an Outpatient Clinic for Teaching and Research in Chest Disease
David H. Law, Nashville

Negative Pressure Breathing
Mortimer E. Bader, New York City

Friday, June 30

10:00 a.m.

Moderator:

Criteria for Diagnosis and Classification of Bronchitis, Asthma and Pulmonary Emphysema
Julius L. Wilson, New York City

Demonstrators:

Pulmonary Physiology in Heart Surgery
Warren Taylor, Boston

Clinical Evaluation of Pulmonary Emphysema
H. F. Helmholz, Jr., Rochester, Minn.

Office Testing of Pulmonary Function
Glenn E. Horton, Memphis

ALTERNATES:

George R. Meneely, Nashville
W. Clark Cooper, Washington, D. C.
John B. Hickam, Indianapolis
Frank Princi, Cincinnati
Maurice S. Segal, Boston
Peter A. Theodos, Philadelphia

NEW YORK STATE CHAPTER MEETING
AMERICAN COLLEGE OF CHEST PHYSICIANS

Friday, June 23, 8:00 p. m. — Commodore Hotel, New York City

Eleventh Annual Howard Lilienthal Lecture

Introduction by: Andrew L. Banyai, Chicago, Clinical Professor of Medicine Emeritus, Marquette University (Milwaukee)

"Tuberculosis in a Changing World"

Herman E. Hilleboe, Albany, Commissioner of Health, State of New York

All physicians are cordially invited to attend

Friday, June 23

9:00 a.m.—Open Forum

Unsolved Problems in the Prevention and Treatment of Tuberculosis

Sponsored by the Committees on Tuberculosis, Chemotherapy and Antibiotics, and Non-Surgical and Drug Therapy

Moderator:

Karl H. Pfuetze, Chicago, Medical Director and Superintendent, Chicago State Tuberculosis Sanitarium

Panel:

Edward T. Blomquist, Washington, D. C., Medical Director, Chief, Tuberculosis Program, Bureau of State Services, Public Health Service, Department of Health, Education, and Welfare

Sidney H. Dressler, Denver, Chief of Staff, National Jewish Hospital

W. Leonard Howard, Northville, Michigan, Superintendent, Wm. H. Maybury Sanatorium

Edith M. Lincoln, New York City, Adjunct Professor of Pediatrics, New York University

Questions and Answers from the Floor

Physicians concerned with the eradication of tuberculosis will find this open forum stimulating and informative. All members of the College are invited to attend.

10:30 a.m.—Open Forum

Sponsored by the Council and Committee on Undergraduate Medical Education

The Chest Conference Approach to the Teaching of Diseases of the Chest

BY MEMBERS OF THE STAFF OF THE UNIVERSITY OF BUFFALO SCHOOL OF MEDICINE AND THE BUFFALO GENERAL HOSPITAL

Moderator:

Theodore H. Noehren, Assistant Professor of Medicine

Panel:

Surgeon	**Richard H. Adler,** Assistant Professor of Surgery
Allergist	**Carl E. Arbesman,** Assistant Clinical Professor of Medicine
Angiologist	**Ivan L. Bunnell,** Assistant Professor of Medicine
Radiologist	**Gordon J. Culver,** Clinical Professor of Radiology
Physiologist-Internist	**Howard G. Dayman,** Assistant Professor of Medicine
Cardiologist	**David G. Greene,** Professor of Clinical Research in Cardiovascular Disease
Internist	**Miller H. Schuck,** Associate in Medicine

This year's session on the teaching of diseases of the chest will be devoted to a demonstration and discussion of the integrated chest conference as a teaching method. There will be case presentations, both known and unknown, as a demonstration of a variety of approaches and as a basis of discussion of the various methods in which these teaching conferences can be organized fruitfully. Teachers from other schools have been invited to participate and all physicians interested in teaching are invited to attend this instructive forum and join the discussion.

ADMINISTRATIVE AND SOCIAL FUNCTIONS

Thursday, June 22

4:00 p.m.—Committee on College Bylaws

4:30 p.m.—Committee on Nominations

5:00 p.m.—Editorial Board (*Diseases of the Chest*)

8:00 p.m.—Executive Council

Friday, June 23

8:30 a.m.—Council Meetings
(Undergraduate and Postgraduate Medical Education, Cardiovascular and Pulmonary Research, and Hospitals and Public Health)

9:00 a.m.—Examinations for Fellowship

12:00 noon—ROUND TABLE LUNCHEON MEETINGS (See page 344)

12:00 noon—JOINT LUNCHEON MEETING
Board of Governors and Board of Regents

2:00 p.m.—OPEN ADMINISTRATIVE MEETING

2:00 p.m.—Examinations for Fellowship

2:30 p.m.—**Meetings of all College Committees**

4:00 p.m.—Meeting, Board of Examiners

4:30 p.m.—Council Meetings
(Undergraduate and Postgraduate Medical Education, Cardiovascular and Pulmonary Research, and Hospitals and Public Health)

5:00 p.m.—Joint Meeting
(Members of the National and State Committees on Membership, College Chapter Officials, and members of the Committee on Liaison with State and County Medical Societies)

Saturday, June 24

5:00 p.m.—Chapter Meetings

5:00 p.m.—Committee on Emphysema

Sunday, June 25

6:00 p.m.—ANNUAL CONVOCATION

Presiding Officers:
M. Jay Flipse, Miami, President
Arthur M. Olsen, Rochester, Chairman, Board of Regents

Sixth Annual Louis Mark Lecture
"The Doctor in Court"
Judge Warren E. Burger, United States Court of Appeals, Washington, D. C.

Conferring of Honorary Fellowships and Fellowships

Address
Hollis E. Johnson, Nashville, President-Elect
American College of Chest Physicians

7:00 p.m.—Cocktail Party

7:30 p.m.—ANNUAL PRESIDENTS' BANQUET
Strictly social — no speeches

9:30 p.m.—ANNUAL COLLEGE DANCE
Sponsored by the New York State Chapter of the College

The Arthur Murray Dancers will present a "Champagne Hour"

Monday, June 26

7:30 a.m.—Past-Presidents' Breakfast

LADIES ACTIVITIES

Saturday, June 24

12:30 p. m. — Fashion Luncheon—Hotel Pierre, Cotillion Room
Fifth Avenue at 61st Street
"Designer Fashions in Hats"
Mrs. Arthur Murray, known as Kathryn Murray to legions of TV viewers, will m.c. the fashion show.
Hats by Vincent-Harmik; Furs by Cali; Pianist, Mr. Forrest Perrin; Fashion Coordinator, Peggy Cantor
A Lanvin perfume gift for every guest. Door prizes will include beauty treatments at Fifth Avenue salons including Helena Rubenstein, Antoine's, the Ritz Tower; beauty gifts from Elizabeth Arden, Dorothy Gray, and others.

Due to the limited seating capacity, reservations for the fashion luncheon must be made in advance. Requests for reservations will be accepted in the order received. (Tickets $6.00 each)

Sunday, June 25

6:00 p. m. — College Convocation
7:00 p. m. — Cocktail Party
7:30 p. m. — Annual Presidents' Banquet
Strictly a social event — no speeches!
9:00 p. m. — College Dance
Sponsored by the New York State Chapter of the College
The Arthur Murray Dancers will present a "Champagne Hour"

Monday, June 26

8:00 p. m. — **"Fun with the Mind"** — Commodore Hotel
A unique exhibition of mind-reading and hypnosis by Franz Polgar, known as the world's foremost hypnotist. Dr. Polgar's frequent TV appearances include the Garry Moore Show, Mike Wallace, Arthur Godfrey and Jack Paar.

Tickets for the "Fun with the Mind" show will be available to ladies only, at time of registration.

TECHNICAL EXHIBITORS

Barnes-Hind Pasna Company
Sunnyvale, California

Warren E. Collins, Inc.
Boston, Massachusetts

Consolidated Midland Corporation
Katonah, New York

F. A. Davis Company
Philadelphia, Pennsylvania

DeVilbiss Company
Somerset, Pennsylvania

Dumas-Wilson & Company
St. Louis, Missouri

Dynamics Corporation of America
New York, New York

J. H. Emerson Company
Cambridge, Massachusetts

Encyclopedia Britannica
New York, New York

E. Fougera and Company, Inc.
Hicksville, New York

Grune & Stratton, Inc.
New York, New York

Instrumentation Associates
New York, New York

Jones Medical Instrument Company
Chicago, Illinois

Eli Lilly and Company
Indianapolis, Indiana

Linde Company
New York, New York

Thomas J. Mahon Company
Englewood, New Jersey

Merck Sharp & Dohme
Philadelphia, Pennsylvania

Micro X-ray Recorder Company
Chicago, Illinois

Mine Safety Appliances Company
Pittsburgh, Pennsylvania

Mist O_2 Gen Equipment Company
Oakland, California

J. J. Monaghan Company
Denver, Colorado

National Cylinder Gas Company
Chicago, Illinois

North American Philips Company
New York, New York

Ohio Chemical & Surgical Equipment Co.
Madison, Wisconsin

Oxy-Lyfe Corporation
Chicago, Illinois

The Panray Corp.
Englewood, New Jersey

George P. Pilling & Son Company
Philadelphia, Pennsylvania

Puritan Compressed Gas Corporation
Kansas City, Missouri

W. B. Saunders Company
Philadelphia, Pennsylvania

Sherman Laboratories
Detroit, Michigan

U. S. Vitamin & Pharmaceutical Corp.
New York, New York

Vaponefrin Company
New York, New York

Wampole Laboratories
Stamford, Connecticut

Winthrop Laboratories
New York, New York

HOTEL RESERVATION FORM

27th Annual Meeting
AMERICAN COLLEGE OF CHEST PHYSICIANS
Hotel Commodore, New York City
June 22-26, 1961

Annual Meeting, American Medical Association
New York Coliseum, June 25-30, 1961

In order to be assured of desired accommodations for the June meetings, please mail this reservation form **at once** to the Hotel Commodore. Please be sure to indicate exact arrival and departure dates.

You will receive a confirmation of your reservation from the Commodore Hotel at an early date.

Reservation Department
HOTEL COMMODORE
42nd St. at Lexington Ave.
New York 17, N. Y.

I am planning to attend the 27th ANNUAL MEETING OF THE AMERICAN COLLEGE OF CHEST PHYSICIANS. Please confirm the following reservations to me:

Singles	$10.00 $16.00	$11.50 $17.00	$12.50 $18.00	$13.50 $19.00	$14.00	$15.00
Doubles	$17.00	$18.00	$19.00	$20.00	$21.00	
Twins	$18.00 $23.00	$19.00 $24.00	$19.50 $25.00	$20.00	$21.00	$22.00
Parlor & Bedroom	$23.00	$25.00	$35.00	$55.00		
Studio Parlor	$20.00	$25.00	$30.00			

PLEASE CIRCLE ROOM RATE DESIRED

Name_____

Address_____

City/State_____

Accompanied by_____

Arrival Date_____ Departure Date_____

Remarks_____

ADVANCE REGISTRATION

There is no registration fee for members

The registration fee for non-members is $25.00

(FEE INCLUDES ATTENDANCE AT THE POSTGRADUATE SEMINARS)

By completing this form and returning it at once to the Executive Offices of the College, you will avoid having to stand in line at the Registration Desk at the annual meeting in New York City. Your badge and program, as well as luncheon, banquet and seminar tickets, will be prepared in advance and will be awaiting your arrival at the Commodore Hotel. Please complete both sides of this Advance Registration Form and mail promptly. Thank you.

Non-members must enclose a registration fee of $25.00 with this form. Please make checks payable to the **American College of Chest Physicians.**

Return this form to: American College of Chest Physicians
112 East Chestnut Street
Chicago 11, Illinois

FOR HOTEL RESERVATIONS PLEASE COMPLETE FORM ON PAGE 354

Advance Registration Form

_____ Member _____ Non-member

Please Print

Name _____

Address _____

City/State _____

Accompanied by _____

Hotel _____ Arrival date _____ Departure _____

Reservation Form—Annual Presidents' Banquet

Sunday, June 25, 1961
Commodore Hotel, New York City

Cocktails — Dinner — Dancing
Tickets: $11.50 each

I wish to reserve _____ tickets for the Annual Presidents' Banquet, Sunday, June 25, 7:00 p.m. (price includes cocktails, dinner and dancing).

ALL SEATS RESERVED

RESERVATION FORM

Annual Presidents' Banquet

This year the Annual Presidents' Banquet, as well as the Convocation, Cocktail Party and College Dance, will be held on Sunday night, June 25. All places at the banquet are reserved and tables will be assigned in the order requests are received. Please order your tickets at once so that you may be assigned your table early. Tables for ten may be reserved upon receipt of payment for tickets and the names of the persons in the party. A reservation form may be found on the reverse side of this page.

Postgraduate Seminars

The program of postgraduate seminars appears on pages 327 and 328 of this issue of the journal. There is a tuition fee of $7.50 for each seminar. Registration for the postgraduate seminars is limited and reservations will be accepted in the order received. Please use the reservation form on this page and submit promptly to the Executive Offices of the College.

Round Table Luncheons

Round table luncheon discussions will be held on Friday, Saturday and Sunday, June 23, 24 and 25, at the Commodore Hotel, and on Monday, June 26 at the Park Sheraton Hotel, which is a joint presentation of the College and the American Medical Association. The subjects and discussion leaders may be found on pages 344 to 347 of this journal. Tickets for the round table discussions are usually sold out in advance of the meeting and members are urged to make their reservations at once. Please fill out the reservation form at the bottom of this page.

Postgraduate Seminars, Thursday, June 22

$7.50 each for members
Included in the $25.00 Registration Fee for Non-members

Morning Sessions (9 a. m. - 12 noon)		**Afternoon Sessions (2 p. m. - 5 p. m.)**	
First Choice	AM_____	First Choice	PM_____
Second Choice	AM_____	Second Choice	PM_____

Please indicate choice by number as listed in program

Round Table Luncheons

TICKETS: $5.75 each

	Friday, June 23	**Saturday, June 24**	**Sunday, June 25**	**Monday, June 26**
First Choice	A_____	B_____	C_____	D_____
Second Choice	A_____	B_____	C_____	D_____
Third Choice	A_____	B_____	C_____	D_____

Please indicate choice by number as listed in program

I am enclosing my check in the amount of $_____ for reservations as indicated on this form.

Please make checks payable to **American College of Chest Physicians**